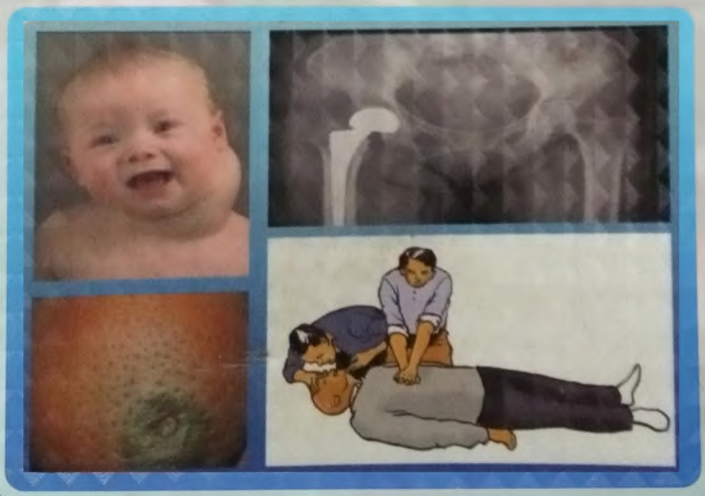
Illustrated Surgery For Undergraduates

By Staff members of General Surgery department
Faculty of Medicine - Zagazig University

Editors

Prof. **Ahmad Kamal** Prof. **Wael Lotfy**



Part 1

First Edition 2018

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Part I

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Preface

Hopefully, this book will help the undergraduate students to understand basic surgery and provide them with an easily revised reference in the pre-examination period .

Although this book was mainly written for undergraduates, yet, we hope that it will provide the postgraduates & general practitioners a brief compact textbook in surgery.

We find a great pleasure in expressing our indebtedness & gratitude to our professors & colleagues in general surgery department Zagazig University who revised this book , for their precious advice & kind notes especially **Prof. Ahmad Kamal** and **Prof. Wael Lotfy**; the editors of this book who did a great effort and suffered much during the preparation of this edition and our deep and great thanks to **Dr. Yasser Nasr** who drew the beautiful diagrams and pictures of this book

Also, we feel thankful to our students who through their fine but wonderful remarks , share greatly in the production of this book.

Finally we should not forget the great efforts and wonderful patients of **Miss Sahar Ismail** in **Al-Esraa company for computer researches** in typing the manuscript of this book.

The Head Of General Surgery Department

Prof. Khaled Safwat

Dedicated to

The soul of our great teacher

Professor :

Mohamed Abdel Lateef

Table of contents

Chapter I....Trauma

- Wounds 1
- Haemorrhage & blood transfusion 10
- Haemostasis 17
- Shock 25
- Crush syndrome 34
- Cardiac arrest 35
- Burn 37
- Skin graft & flaps 43
- Multiple injuried patient 47

Chapter II...Surgical nutrition

- Fluids, electrolytes and acid–base Balance 51
- Malnutrition in surgery 64
- Nutritional support of surgical patients 65

Chapter III .....Infection

* Definition 67
* Specific infection 68
* Non Specific Infections 78
* Blood infections 85
* Hospital-acquired infections 86
* Surgical Site Infection 88

Chapter IV ..... Organ Transplantation

* Principals of organ transplantation 91
* Renal transplantation 93
* Liver transplantation 94
* Pancreatic transplantation 95
* Small bowel transplantation 96
* Heart transplantation 97
* Lung transplantation 98
* Bone marrow transplantation 98
* Endocrine cell transplantation 98

Chapter V ....General Organs

- The Skin & Subcutaneous Tissues 99
- The Muscles, Tendons And Related Structures 123
- The Veins 129
- The Arteries 147
- The Lymphatic System 170
- The Nerves 184
- The Bones 201

Chapeter VI .....Hand & foot surgery

- Hand injury 271
- Hand infection 274
- Foot infections 282
- Chronic leg ulcers 285

Chapter VII .....Head & Neck surgery

- The Scalp, Skull & Brain 287
- Face lip & palate 311
- The Mouth, cheek & Tongue 318
- Gum, Teeth & Jaws 324
- The Major Salivary glands 331
- Neck Surgery 344
- The thyroid gland 358
- Parathyroid glands 390

Chapter VIII ....Breast surgery

- Embryology & anatomy of the breast 395
- Histology & physiology of the breast 398
- Congenital Anomalies 399
- Traumatic Conditions 399
- Inflammatory Conditions 401
- Mammary Dysplasia 406
- Tumours of the Breast 408
- Hard Swellings of the breast 426
- Cysts of the breast 426
- Discharge per nipple 427
- Diseases of the male breast 428

Chapter I

Trauma

WOUNDS

Definition:

Loss of tissue continuity caused by mechanical trauma.

Aetiology = mechanical trauma.

1- Blunt trauma: → Direct compression of tissues leads to lacerations.
→ Acceleration- deceleration injury.

2- Penetrating trauma: → Stabbing: trauma along the track only.
→ Firearm injury: trauma along the track plus shock waves spread out of the track.

3- Blast injury: → Blast waves → shearing of the tissues.
→ Produced missiles → penetrating injury.

Pathology:

Classification:

I- Medicolegal classification

A-Closed wounds (partially or completely intact skin).

1-Abrasions (سحجات): Degloving of the superficial layers of the skin due to friction against rough surface.

Abrasion

2-Contusions (كدمات): extravasation of blood in the tissue planes due to injury of small blood vessels by blunt trauma with intact skin (ecchymosis). Due to disintegration of extravasated blood, the area is at first red then within few days, it becomes yellow and later becomes greenish and lastly fades out.

Contusion

3-Haematoma(تجمع دموى): extravasation of blood in the deep tissues due to injury of a sizable blood vessel. The fate of haematoma may be

1. -Absorption.
2. -Encapsulation (cyst).
3. - Organification (fibrosis).
4. - Calcification.
5. -Infection (abscess).
6. -False aneurysm.

Haematoma

B-Open wounds: (loss of skin continuity).

1-Incised wound (جرح قطعى):

-It is caused by sharp pointed objects.

-It is longer than deeper.

-The edges are clean cut & the tissue damage is minimal.

-It bleeds profusely (clean-cut vessels).

-Contamination and sepsis are minimal.

Incised wound

2-Lacerated wound (جرح فتكى):

-It is caused by blunt trauma.

-The edges are crushed and tissue damage is marked.

-Little bleeding (crushed vessels).

-Sepsis is more liable to occur.

3-Penetrating wound (جرح نافذ):

-It is caused by penetrating trauma.

- It is deeper than longer.

-Internal organs may be injured with possible internal haemorrhage. It includes:

Penetrating wound

- Puncture wound: It has an inlet only.
- Perforating wound: It has an inlet and exit e.g. bullet or transfixing stabs.

4- Crushed wounds (Shell fragment wound):

They are compound wounds which combine lacerations, penetration and fractures.

II-Therapeutic classification.

Wounds are classified into:

1-***Clean wounds***: Wounds within 6 hours, with no gross dirt or devitilized tissue, in clean areas, which have not been subjected to unclean manipulation. Infection rate is 2%.

2- **Clean contaminated wounds:** they are similar to clean wounds but a hollow organ was entered without marked spillage of contents. Infection rate is 4%

2-***Contaminated wounds***: Wounds after 6 hours, containing dirt or devitalized tissue, in contaminated areas (e.g. perineum) or subjected to unclean manipulation or a hollow organ was entered with marked spillage of contents. Infection rate is 7 – 10 %

3- ***Dirty & Infected wounds:*** They show signs of inflammation and discharge pus.

Healing of wounds

***Definition:** It is replacement of lost tissue by granulation tissue that matures into scar.

***Phases of healing:**

1-Substrate (inflammatory) phase: injury induces inflammatory reaction that remains for 3 to 5 days and calls for fibroblasts.

2-Proliferative (fibroplasia & angiogenesis) phase: There is increase in the number of fibroblasts, which lay collagen fibres that form a meshwork holding the wound edges together. Also new capillaries sprout towards the injured area and fuse with those coming from the other side of the wound It extends for several weeks, during which the wound gains more and more tensile strength.

3-Resorptive phases: in this stage, the fibroblasts disappear and excess collagen is removed by phagocytes with maturation of the wound into scar tissue.

Forms of wounds healing

1-First intention: In clean wounds sutured early with minimal gapping, the epithelium crosses the gap first then granulation tissue obliterates the gap. They heal leaving linear *cosmetic* scar.

2-Second intention: Infected or gapped wounds, left open, the granulation tissue fills the gap from the bottom first then the epithelium creeps over it. They heal leaving *ugly* scar.

3-Third intention: Contaminated or infected wounds left opened to granulate till infection subsides ,then sutured with delayed primary or secondary sutures to heal with linear scar.

Sutured clean wound

Primary intention

Infected wound

Secondary intention

Factors affecting wound healing

A-General factors: -

1-Diet:

a) Hypoproteinaemia: It leads to deficient collagen and ground substance (glycoprotein).

b) Hypovitaminosis: especially Vit. C (protocollagen) and Vit. A (epithelium).

c) Minerals: - Ca, Zinc & Cu help wound healing.

2-Diseases:

a) Diabetes mellitus delays wound healing and invites infection.

b) Uraemia, jaundice, liver cell failure & late malignancy delay wound healing.

3-Drugs:

Corticosteriods for long course delay wound healing.

4-Senility:

Wound healing is bad in old age. They have negative nitrogen balance.

5-Heat:

Wound healing is better in hot weather.

6-Humidity:

Humidity accelerates wound healing, but it predisposes to infection.

B-Local factors:

1-Ischaemia:

Poor vascularity and hypoxia hinder wound healing.

2-Infection:

Infection is the strongest enemy for healing.

3-Irradiation:

It interferes with wound healing as it inhibits cell division and causes local ischaemia.

4-Foreign bodies and tissue debris:

They are good media for infection.

5-Frequent mobility and tension:

Shearing movements and tension of wound edges interfere with healing

6-Fixation to bony surfaces:

Adherence to bony surface (e.g. shin of the tibia) prevents contraction of wound edges

Complications:

A) General complications

1- Shock: neurogenic or haemorrhagic.

2- Crush syndrome (see later).

B- Local complications

1-Infection: Pyogenic and specific.

2- Seroma and haematoma

3-Injury of important structures e.g. vessels, nerves & viscera.

4-Gangrene: vascular & infective.

5- Wound dehiscence.

6- Complications of wound healing

a- malignant change (*Marjolin's ulcer*).

b) Sinus & fistula.

c- Contracted scar.

d) Keloid & hypertrophied scar.

e) Disfigurement

f) Lymphoedema in circumferential wound.

Hypertophied scar

Keloid

contracted scar

Treatment:

I- Primary survey: to detect what is killing the patient.

II- Resuscitation (A,B,C,D,E): to manage what is killing the patient.

III- Secondary survey: to detect other possible injuries.

IV- Definitive management:

A) Clean wounds:

1-Wash the wound with saline and remove any foreign bodies & dead tissues.

2-All deep structures should be dealt with immediately.

3-Close the wound with primary sutures hoping to heal with first intention.

B) Contaminated wounds:

1- Irrigate, irrigate, irrigate to ensure that all particulate matter had been removed.

2- Remove all foreign bodies and devitilized tissues.

3- Important structures only (e.g. important vessels) are dealt with immediately, but tendons & nerves are only marked for later repair.

4- Cover vascular anastomosis with muscle or fascia but otherwise, let the wound open for repeated dressing until it becomes clean then close it with delayed primary sutures.

C) Infected wounds

1- Remove all sutures, open all pockets of pus and remove all necrotic tissues.

2- Wash with antiseptic solution.

3- Let the wound open for repeated dressing until it becomes clean:

- If the gap is small, do secondary sutures.
- If the gap is large, apply skin graft (delayed grafting).

Winnett Orr technique = *closed plaster technique*: It was used for severely infected lacerated wounds of extremities especially if associated with fractures. It entails:

*Wide drainage of the wound. * Bulky dressing. *Complete plaster cast.

- Inspite of marked soaking & foul odour, the plaster is changed every 3-6 weeks till healing except in

-High fever -Severe pain -Evidence of secondary hage.

Advantages:

1-Immobilization helps healing, reduces pain and improves toxaemia (↓↓ lymph flow).

2-Infrequent dressing decreases cross infection, patient anxiety, and trauma to the wound.

3-It spares time, money and effort.

Disadvantages:

1- Very offensive odour that may cause psychological upsets to the patients and attendants.

2- It predisposes to anaerobic infection.

D) Penetrating wounds:

1- Widening of the wound & exploration of deep structures.

2- Dealing with injured structures.

E) Closed wounds:

1) *Abrasion:*

It should be managed as superficial burns with closed dressing (Vaseline gauze , bulky dressing and crépé bandage with antibiotics.

2) *Contusion:*

Fomentation, (cold in the 1st 24 hours then hot in the next days).

3) Haematoma

-Small haematoma: Fomentation as before + bandage + antibiotics. Usually it is absorbed

-Large haematoma: Aspiration or evacuation + bandage + antibiotics.

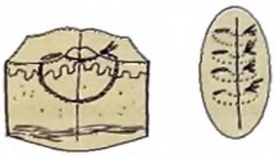
-Increasing haematoma: incision + dealing with the bleeder (e.g. ligation).

Wound closure

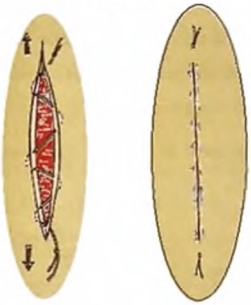
I- Methods of suturing

Sutures may be interrupted, continuous, vertical mattress, horizontal mattress or subcuticular.

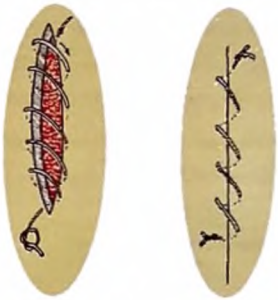
Interrupted sutures



vertical mattress sutures



Subcuticular sutures



Continuous sutures

Removal of sutures:

The sutures should be removed when adequate wound healing is depicted that prevents dehiscence after removal of sutures. Early premature removal of sutures leads to wound gapping while neglected delayed removal of sutures leads to bad cosmoses and infection or sinus formation (stitch sinus)

II- Suture material

There is wide variety of suture materials which can be classified into:

I- Absorbable:

A-Natural sutures (absorbed by phagocytosis):

1- Catgut sutures: It is monofilament derived from cattle and goat gut. It is rapidly absorbable. It may be plain or chromic. It is no more used in developed countries.

B- Synthetic sutures (absorbed by hydrolysis):

1- Polyglycolic acid (Dexon): It is braided (مضفر) multifilament.

2- Polyglactan (Vicryl): It is braided multifilament, the most widely used absorbable sutures

3- Polydioxanone (PDS). It is monofilament delayed absorbable suture

Absorbable sutures lose their strength with time.

II- Non-absorbable sutures:

A- Natural sutures:

1- Silk sutures: it is braided multifilament.

2- Linen:

B- Synthetic Sutures:

1- Nylon (Ethilon): it is monofilament

2- Polyprolene (prolene): it is monofilament and the most widely used non-absorbable sutures

3- Expanded polytetrafluoroethylene (ePTFE) (Gortex): it is monofilament inert but expensive

4- Stainless steel wire: it is used in closure of sternotomy in open heart surgery.

Non-absorbable suture retain their tensile strength forever and are used in closure of abdominal incisions and repair of hernias or in closure of skin wounds

Suture Types	Knot pull strength	Knot security	Handling	Tissue reactivity	In vivo strength loss
Catgut	Poor	Poor (plain) Fair (chromic)	Fair	High	7-10 days (plain), 21-28 days (chromic)
Silk	Fair	Good	Very good	High	1 year
Polypropylene	Fair	Poor	Poor	Low	Indefinite
Polyamide	Fair	Fair	Good	Low	1.5 – 2.5% /year
Stainless steel	High	Good	Poor	Low	Indefinite
PGA/PLLA	Good	Fair -good	Good	Low	10 days – 4 weeks
Polydioxanone	Fair -good	Poor-fair	Fair-good	Low	10 days – 6 weeks

Suture size

Suture sizes are standardized.

- The more zeros in the number, the smaller the size of strand (سُمك الخيط). (2/0, 5/0 ... etc)
- The smaller the size of the suture, the less tensile strength.

Surgical needles

Ideal surgical needles: 1- Stainless steel 2- slim but strong
3- stable with needle holder. 4- With minimal trauma
5- Penetrate tough tissues securely. 6- Preserve delicate tissues

Basic needle design: -it has a swage (*suture place*), body (*grasped by needle holder*) and tip (*the first penetrating part*).

-Needles may be straight or curved.

-The curve may be 1/4, 3/8, 1/2, or 5/8 circle

-the length may be 6,8,12 .....50 mm

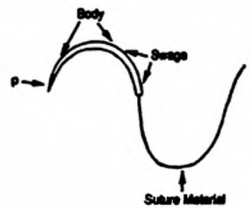
-The cross section may be

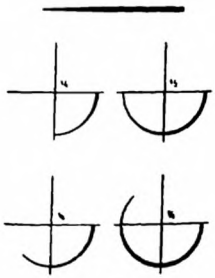
Round (smooth round cut section), or

Cutting (triangular cut section)

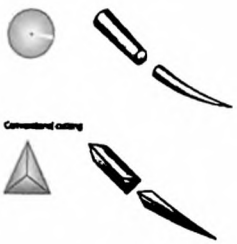
-The tip may be blunt or tapering

-the surgeon selection depends on nature of tissues and space available for suturing





Needle curve



cross section

Definition

Extravasation of blood.

Aetiology:

I-Trauma: Accidental, suicidal or surgical intervention.

II-Pathological:

1-Bleeding tendency: platelets, coagulation or vascular defects.

2-Inflammation: Inflammation may erode blood vessels e.g. peptic and typhoid ulcers.

3- Degenerative: rupture cysts and aneurysms.

4- Neoplastic: malignant infiltration of blood vessels.

Pathology

Types:

I-According to the site:

A-External haemorrhage: visible bleeding from wounds or from body orifices .

B-Internal haemorrhage: bleeding in serous cavities e.g. peritoneum, pleura or pericardium.

C-Interstitial haemorrhage: bleeding in tissue planes e.g. intramuscular haematoma

II-According to the source of bleeding :

A) Arterial: pulsating jets of bright red blood more from the proximal end and can be stopped by firm compression or ligation of the artery.

B) Venous: steady flow of dark red blood more from the distal end, and can be stopped by light compression or just elevation of the bleeding limb.

C) Capillary: diffuse ooze of bright red blood from multiple points & stopped by compression.

III-According to the time in relation to trauma:

A) Primary haemorrhage: immediate at the time of trauma.

B) Reactionary haemorrhage: within 24-48 hours after trauma due to:

*Rise of blood pressure. *Slipped ligature. *Dislodged blood clot.

C) Secondary hemorrhages: within 2 weeks after trauma due to infection eroding the vessel

Physiological response to haemorrhage

I-General response: See hypovolaemic shock

II-Local responses = Natural arrest of bleeding

1- Vasoconstriction and retraction of the intima of the injured vessel.

2- Formation of platelets plug.

3- Activation of natural clotting mechanism.

4- Lowering of blood pressure.

5- Compressing action of haematoma and tissue tension.

Complications: -

1-Shock: hypovolaemic shock in severe haemorrhage.

2-Anaemia: haemorrhagic in marked bl. loss or iron deficiency anaemia in chronic mild bl. loss.

3-Complications of haematoma, see before.

Clinical pictures:

The clinical manifestations vary according to the amount of blood loss:

Item	Class I	Class II	Class III	Class IV
Blood loss	< 750ml	750 – 1500 ml	1500 –2000 ml	> 2000 ml
Mental status	Normal to anxious	Anxious to restless	Restless to drowsy	Drowsy to comatosed
Pulse	<100 b/ min	100 – 120 b/ min	120 –140 b/ min	>140 b/ min
Bl. pressure	Normal	normal	low	low
Resp. rate	14 –20 /min	20 –30 /min	30 –40 /min	> 40 /min
Skin	Normal	Pale & cool	Pale & cooler	Pale & very cold
Urine output	> 30 ml /h	20 – 30 ml /h	10 –20 ml /h	< 10 ml /h

Treatment:

I-First Aid treatment

A-Stop external bleeding: by 3Ps

1) Position: elevation of the bleeding limb.

2) Packing the wound with sterile dressing.

3) Pressure applied through:

a) Bandage over the wound.

b) Inflating a sphygmomanometer cuff over the wound.

c) Pneumatic antishock garment (PASG).

d) Balloon tamponade: for bleeding oesophageal varices

e) Tourniquet: It should be avoided. (More limbs were lost than lives saved by the tomiquet)

Complications of tomiquet (if neglected)

i) Gangrene. ii) Crush syndrome. iii) Nerve injury.

B-Resuscitation:

Restoration of the blood volume is of great importance by:

1) Blood transfusion. 2) IV fluids.

II-Surgical arrest of bleeding

A-Primary haemorrhage:

Exploration of the wound and ligation of the unimportant vessels or reconstruction of important ones.

B-Reactionary:

Re-explore the wound and control bleeding by ligation or pack.

C-Secondary haemorrhage:

- Re-explore the wound under umbrella of antibiotic and ligate the bleeding vessel above and below (healthy ends) then resect it in-between **OR** Apply tight pack for 48 hrs.
- Postoperative splint is applied to prevent spread of infection and rebleeding.

Blood transfusion

Collection & storage:

The blood is collected into special bags containing sodium citrate in saline dextrose then stored in 4°C for up to 3 weeks.

Changes occurring during storage:

1-RBCs: 75% of RBCs survive for 3 weeks. After that the rate of haemolysis accelerates.

2-WBCs: 50% of WBCs survive for 1 week

3-Platelets: survive only for 2-5 days.

4-Coagulation factors: -Prothrombin concentration decreases to 75% after 1 week.
-Factor V decreases to 30% after 1 week

5-Potassium: It triples after one week.

Blood components

Product	Obtained by	Storage life	Indications
1-Packed RBCs	Centrifugation of whole blood	21 days	Severe anaemia.
2-Fresh plasma	Removed after centrifugation		
3-Fresh frozen plasma	Rapid freezing of plasma to –40°C	1 year	coagulation factors def.
4-Platelets concentrate	Centrifuge of fresh plasma	1-3 days	Thrombocytopenia.
5- Cryoprecipitate	Warming of the plasma to 4°C → precipitate→ centrifuge→ cooling	1 year	Haemophilia A & hypo-frininoginaemia.
6-Human albumin	Repeated fractionation of plasma	4 years	Hypoalbuminaemia.

Indications of blood transfusion

I-Medical indications:

1-Severe anaemia: → whole blood or better-packed RBCs in extremes of age.

2-Agranulocytosis: → fresh blood.

3-Thrombocytpenia → fresh blood, fresh plasma or better platelets concentrate.

4-Coagulation disorders e.g. haemophilia A, hypofibrinogenaemia & hypoprothrombinaemia.

5-Hypoproteinaemia: fresh plasma or better human albumin.

II- Surgical indications:

1- Hemorrhage.

2- Shock.

3- Replacement of surgical blood loss.

4- Septicemia & toxemia.

5- To prime the heart lung machine.

Precautions of blood transfusion

1-Blood grouping and cross-matching should be cautiously performed to avoid incompatibility.

2-Banked blood stored at 4°C should be warmed gradually to room temperature before transfusion to avoid hypothermia & arrhythmia.

3-Blood must be transfused separately through its special infusion set containing special filter.

4-No medications should be added to bl. & fluids given in contact with bl. should be free of Ca^{++}.

5-We shouldn't transfuse 2 units of blood at the same time.Because if reactions occur, we can't know which of them causes the reaction and both units should be discarded.

Complications of blood transfusion

Although it is usually life saving, yet it may lead to fatal complications.

1-Haemolytic reaction, (Incompatibility).

Definition: It is an immune reaction between the recipient's antibodies and donor's RBCs that causes their agglutination and haemolysis.

Aetiology: It is due to ABO, Rh (second transfusion) or rarely other blood groups incompatibility. It is usually due to an error in labeling or cross matching.

Pathology: -Agglutinated RBCs will block the microcirculation → tissue ischaemia.

-Haemoglobin released from haemolysed RBCs precipitates in renal tubules as acid haematin causing acute tubular necrosis and anuria

-Agglutinated RBCs may induce DIC leading to bleeding tendency.

Complications:

1-Shock & hypotension.

2- Acute renal failure.

3-Haemolytic jaundice.

Clinical picture:

a) Anaesthetized & unconscious patient:

1) Unexplained hypotension & tachycardia.

2) Fever and flushing.

3) Undue bleeding from the wound and haemoglobinuria in the catheter.

b) In conscious patients: There are in addition

1) Chest tightness.

2) Headache, nausea & vomiting.

3) Loin pain.

Treatment:

"The diagnosis should be clinically made very rapidly "

1- Stop blood transfusion immediately.

2- IV. fluids (Ringer lactate) to compat hypotension & acidosis and increase renal perfusion.

3- Corticosteroids to combat shock and minimize the immune reaction.

4- Forced diuresis by mannitol to wash acid haematin.

5- Alkalization of urine to wash acid haematin.

Prognosis: About 40% mortality rate.

2-Pyrogenic reaction: (the commonest reaction)

Aetiology: It is due to transfusion of pyrogenic substance e.g. bacteria, donner's WBCs, platelets or pyrogens in the transfusion set.

Clinical picture: Fever, rigors, headache, nausea & vomiting.

Treatment: Stop transfusion & give antipyretic drugs.

3-Allergic reaction:

Aetiology: Due to transfusion of allergens in the donor's blood .e.g. platelets or WBCs

Clinical picture: -Urticarial rash with itching.

-Rarely, anaphylactic shock and laryngeal edema.

Treatment: -Stop transfusion + antihistaminics + Corticosteroids.

-Subcutaneous adrenaline in laryngeal edema.

4-Toxaemia & septicaemia:

Aetiology: *It* is due to transfusion of blood containg endotoxin of Gram –ve bacteria.

Clinical picture: Septicaemia with fever, rigor and even septic shock.

Treatment: Corticosteroids, antibiotics & antipyretic.

5-Transmission of diseases:

Viral hepatitis, Acquired Immune Deficiency syndrome (AIDS), Malaria, & syphilis.

6-Microembolization :

Aetiology: Microembli (may be platelets aggregates) may block the pulmonary capillaries.

Clinical picture: Chest tightness with acute respiratory distress.

Treatment: It is prevented by using microfilter containing 40-micron mesh in the infusion set.

7-Complications due to massive blood transfusion (5Hs) :

A) Hyperkalaemia:

Aetiology: High K^+ content in the stored bl. due to exhaustion of K^+/Na^+ pump in hypoxic RBCs.

Clinical picture: Arrhythmia that may end in arrest.

Treatment: -Avoid old blood in old patient.

- K^+ losing diuretics.

B) Hypothermia:

Aetiology: It occurs when large amount of cold-banked blood is transfused rapidly.

Clinical picture: that of acidosis, arrhythmia and may be arrest.

Treatment: It is avoided by warming blood before transfusion.

C) Hypervolaemia:

Aetiology: Overloading of the circulation in a short time especially in extremes of age.

Clinical picture: It is that of heart failure and pulmonary oedema.

Treatment: - It is avoided by slow controlled transfusion over 4-6 hours or use of packed RBCs.

- It is treated by diuretics & digitalis.

D) Hypocalcaemia (citrate intoxication):

Aetiology: Citrate in the transfused blood binds the recipient Ca^{++} causing hypocalcaemia.

Clinical picture: It exaggerates the hyperkalaemic effect on the heart leading to cardiac arrest.

E) Haemorrhagic tendency:

Aetiology: 1-Dilutional thrombocytopenia & decreased concentration of the coagulation factors in the transfused blood.

2-Consumption coagulopathy due to disseminated intravascular coagulopathy.

Clinical picture: -Thrombosis leads to ischaemia that may end in gangrene of skin & digits.

- Undue bleeding from incisions and drains.

Treatment -Heparin to suppress the coagulopathy.

-Epsilon amino caproic acid to suppress fibrinolysis but it may induce renal failure.

-Fresh blood transfusion.

8-Complications due to old blood transfusion

a) Post-transfusion jaundice:

Aetiology: Due to diminished survival of old RBCs.

b) Hepatic precoma & coma:

Aetiology Increased amonia content of the old RBCs.

c) Hyperkalaemia

Aetiology: See before

9-Immunosuppresion:

Aetiology: suppression of recipient immunity by the donner's α macroglobulin.

There is high incidence of malignant recurrence in patients receiving blood transfusion during or after radical resection of cancer.

10-Complications of I.V. infusion

a) Thrombophlebitis.

b) Air embolism.

How to minimize the need for homologous blood transfusion?

1-Autologous blood transfusion: The patient who is going to have a major elective surgery donates some units of his blood either immediately pre operatively or few weeks before surgery. This blood is given back to the patient during or after surgery.

N.B. Frozen blood is a new method to store blood for longer period by freezing fresh packed RBCs to –80: –190°C after adding glycerol. This method is ideal for autologous blood transfusion in elective surgery.

2-Per-operative blood salvage: the blood lost in the operation is collected, washed and re-infused to the patient by a special set (cell saver).

-It is not suitable in unclean surgery or surgery for malignancy.

Haemostasis.

Haemostsis is the mechanisms by which the body tries to arrest bleeding after injury to blood vessels

Physiology of haemostasis

I- Primary haemostasis:

It refers to the initial mechanisms to arrest bleeding in the first few minutes, these include.

A- Vascular mechanisms.

1- Constriction of the injured blood vessels: This occurs due to

1- Vasoconstriction which is achieved by circular muscle contraction.

- Vasoconstriction is less effective in the following conditions:-
  1) When the vessel is partially injured because it is prevented from constriction and retraction.
  2) When the vessels are astherosclerotic .
  3) If large veins are injured as they have relatively poor musculosa.

2- Retraction of injured blood vessels.

The longitudinal muscle fibers of the injured vessels also contract leading to

1) Retraction of the blood vessels inside the tissues that press over the vessel obstructing them.
2) As the vessel is shortened, the intima is crumbled inside the lumen occluding it.

B- Platelet plug formation.

When blood vessel endothelium is injured exposing the subendothelial collagen. The platelets rapidly adhere to it and release their mediators. e.g. thromoboxane & others that enhance more platelets aggregation & adhesion so, platelets will form a plug that occludes the hole in the bl. vessel

C-Tamponade:

Tamponading the injured vessel by

1) Resultant haematoma especially in interstial bleeding rather than external or internal haemorrhage.
2) Tissue tension especially after oedema develops.
3) Reflex contraction of nearby injured muscles..

II- Secondary haemostasis

Primary haemostasis may succeed to arrest bleeding but temporary as when vasoconstriction subsides the blood flow will dislodge the platelets plug if not reinforced with fibrin mesh. So, without blood coagulation bleeding would restart again after ½ hour to several hours after the temporary arrest.

- Blood coagulation can be stimulated through two pathways.

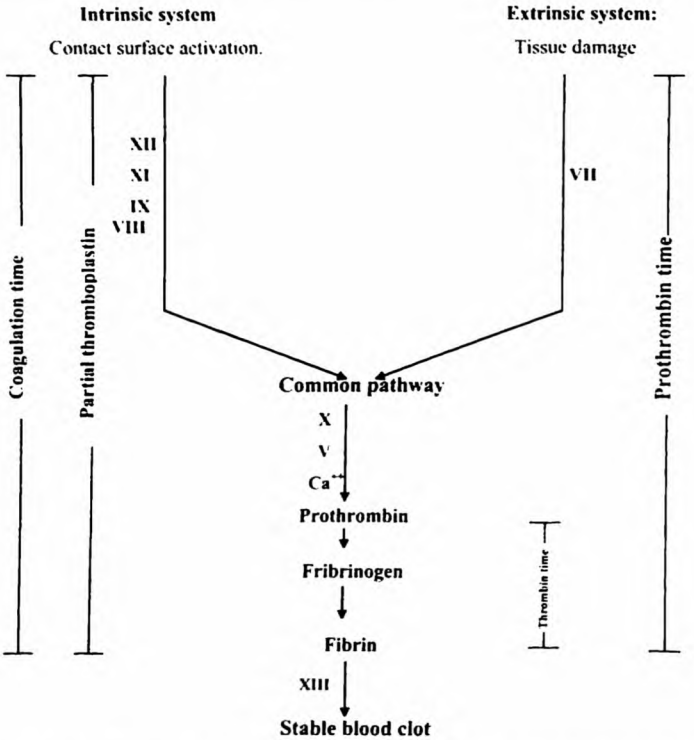
1) Extrinsic system.

It begins with activation factors VII by tissue factor that is released on tissue damage.

2) Intrinsic system:

It is initiated by activation of factor XII on contact with non-endothelial surface.

After initial activation of other coagulation factors takes place in a cascade manner that ultimately end information of fibrin meshwork that entangles blood components forming blood clot or thrombus. Further stabilization of the blood clot through polymerization of fibrin with aid of factor XIII.



As coagulation is self-maintaining procedure. So inhibitors are essential to stop progressive activation of coagulation factors and thus extensive thrombosis. These inhibitors are normally present in blood such as antithrombin III, protein C & S. Deficiency of these factors may cause spontaneous thrombosis e.g. DVT. & mesenteric vascular occlusion.

Fibrinolytic system: -

This system is responsible for dissolution of recently formed blood clot obstructing blood vessels to recanalize them after haemostasis is secured. Circulating plasminogen is activated by tissue plasminogen

activator to form plasmin, which digest the blood clot and restore patency of the vessel. The products of fibrin digestion are called fibrin degradation products (FDPs), which have anticoagulant effect through inhibition of fibrin polymerization.

Again plasmin and plasminogen activators are inactivated by circulating substances that maintain an equilibrium between coagulants and anticoagulants.

Disorders of haemostasis (Bleeding tendency)

Causes:

Haemostasis disorders can be classified into disorders of primary haemostasis that lead to purpura & disorders of secondary haemostasis leading to coagulopathy.

I- Primary haemostatic disorder (purpura):

A- Vascular purpura .

1- Scurvy (vitamin C deficiency leads to impairment of collagen synthesis).
2- Henoch Schonlein purpara.
3- Purpura fulminans is major illness .
4- Splinter haemorrhage under nail in subacute bacterial endocarditis.
5- Abdominal rose spot in typhoid fever.
6- Purpura due to viral diseases e.g. smallpox, measles and chicken pox.
7- Purpura in severe septicaemia e.g. Waterhouse– Friderichsen syndrome; H. influenza septicaemia, Poisons & drugs e.g. snake venom and atropine.

B- Purpura due to platelets defect:

[1] Thrombocytopenic purpura:

a) Idiopathic (Primary or autoimmune) thrombocytopenic purpura (ITP.).

- Due to antibodies against platelets sequestrating them in spleen with subsequent destruction.
- Prolonged bleeding with major surgery or trauma occurs. when platelets count is 70,000-50,000 /mm^3.
- Prolonged bleeding after minor surgery or trauma occurs when platelets count is 50,000-20,000/mm^3
- Spontaneous bleeding occurs when platelets count decreases below 20,000/mm^3.

b) Secondary thrombocytopenic purpura.

1) Thrombotic thrombocytopnia.

   Spontaneous thrombosis consumes platelets leading to thrombocytopenic purpura, haemolytic anaemia and neurological manifestations.

2) Thrombocytopenia secondary to blood disease such as leukaemia and aplastic anaemia .
3) Sequestration thrombocytopenia: Thrombocytopnia due to sequestration of platelets in spleen (in hypersplenism) or in extensive haemangiomas in infants.
4) Thrombocytopenia secondary to drugs e.g sedormid, sulphonamide and quinindine .
5) Thrombocytopenia secondary to viral infection e.g. infectious mononucleosis & rubella.

2- Thrombocytopathic purpura:

Purpura may occur with normal platelets count due to defective platelets function as in

1) Renal & hepatic failures.
2) Macroglabulinaemia .
3) Drugs e.g. Asprin, dipyridamole, some cephalosporines and semisynthetic penicillin.

3- Thrombocythaemic purpura:

Excessive platelets count yet with defective function. It is a rare disease called haemorrhagic thrombocythaemia.

II- Secondary haemostasis defect (coagulopathy):

Coagulation defect may occur in

A- Congenital coagulopathy.

[1] Haemophilia A & B.

- They are the most common congenital coagulapathy .
- Haemophilia A is due to deficiency of factor VIII (antihemophilic factor) and inherited as sex linked recessive trait (from female carriers to males).
- Haemophilia B is less common due to deficiency of factor IX (Chrismas factor) .
- Manifestations vary from recurrent attacks of spontaneous haemorrhage in childhood (when the activity of the factor is below 1%) to excessive bleeding only after trauma when the activity is 5-20%.

[2] Von Willebrand disease (vWD)

- It is due to deficiency of vWD factor, which enhance platelets adhesions and acts as carrier for factor VIII preventing its premature destruction.
- It is inherited as an autosomal dominant character but with variable phenotypes:
  - Type I :Quantitative defect in vWD factor
  - Type II: Qualitative defect in vWD factor
  - Type III Almost absent vWD factor

[3] Others:

Such as: Congenital hypoprothrombinaemia or congenital hypofibrinogenaemia .

B Acquired coagulopathy:

[1] Acquired hypoprothrombinaemia:

Due to :

1) Vitamin K deficiency.
2) Hepatic insufficiency.
3) Malbsorption syndrome.
4) Obstructive jaundice.
5) Haemorrhagic disease of newborn.
6) Prolonged use of intestinal antiseptic.

It commonly occurs in conjunction with factor V, VII & X deficiency because they all are Vit. K-dependent factors. Both PT and PTT are prolonged but thrombin time (TT) is normal.

[2] Acquired hypofibrinogenaemia :-

It occurs mainly in

Disseminated intra-vascular coagulation (consumption coagulopathy):

- It occurs due to intense activation of coagulation factors in the circulation leading to wide spread intravscular coagulation with consumption of the coagulation factors.
- This process ends in tissue infraction and coagulopathy respectively.
- The fibrin degradation products (FDPs) are released into the circulation which worsen the coagulopathy as they interfere with fibin polymerization .
- The causes include: -
  1) Intra-uterine retention of dead foetus, abruptio placenta & eclampsia.
  2) Incompatible blood transfusion, thrombotic thrombocytopenic purpura and leukaemia .
  3) Septicaemia severe trauma & fat embolism.
  4) Hypothermia, hypoxia and severe shock.
  5) Metastatic carcinoma especially of prostate, lung and pancreas.

The manifestations include: -

1-Bleeding tendency. Abnormal bleeding from wounds surgical incisions, drains and venopuncture, also bruises, petchiae or mucosal bleeding may occur.

2-Ischaemic manifestations : gangrene of skin, dight or even extremities may occur.

- The PT, PTT and TT all are prolonged with elevated FDPs and reduced platelets count.

Diagnosis

The accurate diagnosis of haemostatic disorder depends on

I- History:

- The age of onset: onset in childhood (e.g. after circumcision) suggest a congenital disorder recent onset suggest an acquired disorder.
- Family history of bleeding: Positive family history in the maternal side only suggests haemophilia (A or B) while positive history in both paternal and maternal sides suggests von Willbrand disease .
- Before any surgical or interventional procedure, we should inquire about any abnormal bleeding (prolonged or excessive) after circumcision, dental extraction, trauma or surgery also about easy bribability & menorrhagia.
- History of liver or renal insufficiency.
- The character of bleeding:
  - Disorders of primary haemostasis are suggested when.
    - The bleeding is superficial e.g. petichae & ecchymosis in addition to mucous membrane bleeding.
    - Bleeding is excessive immediately following trauma or surgery.
    - Bleeding is controlled by local pressure.

- Disorders of secondary haemostasis are suspected when.
  - The bleeding is deep e.g. haemoarthrosis deep haematoma (in addition to easy bruisability).
  - The onset is relatively delayed after trauma or surgery.
  - Local pressure usually fails to control bleeding.
  - Excessive fibrinolysis is suspected when there is multiple bleeding sites.
- History of drug intake e.g. aspirin , sedormid, anticoagulants, sulphonomide and antibioties .

II- Examination:

- General signs of hepatic or renal failure.
- Petichae, ecchymosis or bleeding mucous membranes usually refer to purpura .
- Haemoarthrosis or deep haematoma refers to coagulopathy.
- Hepatomegaly & splenomegaly may point to leukaemia, hypersplenism or lymphoma. Lymph node enlargement is found also in the same conditions.

III- Investigations:

A. Tests for primary haemostasis:

1) *Bleeding time* .It is prolonged in both vascular and platelets dysfunctions.
2) *Platelets count*: Normal = 150,000 – 400,000/mm^3. It is decreased in thrombocytopenic purpura.
3) *Bone marrow picture*. The megakaryocytes are increased in thrombocytopenia due to peripheral cause e.g. destruction or sequestration and markedly decreased due to bone marrow cause like suppression or replacement as in leukaemia.
4) *Platelets function test* (adhesion, release and aggregation) are needed only if the count is normal while the bleeding time is prolonged.

B- Tests for secondary haemostasis:

1- Coagulation time:

It is prolonged in all types of coagulopathy.

2- Prothrombin time (PT).

It measures the time of clotting through the extrinsic and common pathway i.e. factors VII, X, V, II and fibrinogen.

3- Partial thromboplastin time (PTT).

It measures the time of clotting through the intrinsic and common pathway i.e. factors XII, XI, IX, VIII, X, V, II and fibrinogen.

4- The thrombin time (TT).

It measures the time of clotting of blood when thrombin is added. It tests abnormalities of fibrin formation e.g. hypofibrinogenaemia.

5- Measuring FDPs level.

It is increased in DIC and fibrinolytic syndrome.

6- Individual coagulation factor assay.

To determine the deficient factor specifically.

7- Clot stability test: -

Treatment:

I- First aid:

- First of all, all needed laboratory investigations such as PT, PTT, platelets count are ordered before any replacement therapy is allowed
- Fresh blood or fresh frozen plasma is initially given for replacement. 3-4 units of fresh frozen plasma are usually sufficient to correct moderate degree of coagulopathy.
- Crystalloids are not given to correct shock except if badly needed as they dilute clothing factors and platelets.
- Correction of hypothermia, acidosis and hypoxia.

II- Correction of the cause:

After accurate identification of the haemostatic disorder definitive treatment should takes place.

A- Purpura:

1- Platelets transfusion is necessary in platelets disorders to raise the platelets count > 50.000/cmm

2- Vitamin C therapy for scurvy.

B- Coagulopathy:

[1] Haemophilia .

1) Avoid aspirin and other nonsteroidal anti-inflammatory because of their antiplatelets effect.

2) Avoid intramuscular injection as large haematomas may develop.

3) Vaccination against hepatitis is usually required because the patient needs repeated transfusion.

4) Before any surgery, infusion of factor VIII immediate before operation and for 10 days thereafter.

[2] Von Willebrand disease.

- Infusion of intermediate purity factor VIII concentrate (contains both factor VIII and vW factor).

[3] Hypoprothrombinaemia (due to hepatic insufficiency).

1) Vitamin K.daily intramuscular injection for 3 days.

2) Fresh frozen plasma 2-3 units usually replace the deficient coagulation factors and fibrinolysis inhibitors .

3) Desmopressin (0.3 μg/kg) can raise the level of factor VIII and vWF and shortens the bleeding time before any invasive procedure.

4) Antifibrinolytic agents e.g. tranxemic acid are also beneficial.

[4] Disseminated intravascular coagulopathy .

1) Treatment of the cause or predisposing factor to cut through the viscious circle e.g. correction of dehydration or septicaemia .

2) Fresh blood or fresh plasma (3-4) units initially. Also, platelets transfusion is usually necessary (10-12 packs may be needed initially).
3) Heparin to stop the thrombotic tendency in a dose of 10 units /Kg continuous IV infusion but it is still a matter of controversy.
4) Epsilon Amino caproic acid (EACA) was routinely given for treatment of DIC however; bilateral renal necrosis may follow its administration.

Surgical Haemostasis

Haemostasis during surgery is achieved by various methods

1) Ligation :

First, the bleeder is clamped with an artery forceps then a ligature using surgical suture is tied around it which the forceps is released .

2) Under-running suture :

Using surgical needle and threads , the bleeder is under run by passing the needle in the tissue under it. then the suture is tied firmly over it .

3) Coagulation :

Thermal cauterization of the bleeder can arrest bleeding . various methods are in use .

A- electro cautery : used to stop bleeding from blood vessels 3mm in diameter or less it is either :-

*Monopolar diathermy : in which the surgeon uses the active electrode with small tip to concentrate heating at the bleeding site and from which , electric current passes in the body to reach an exit through broad grounding pad attached to a wide area of the body surface .

*Bipolar diathermy : in which ,the surgeon grasps the bleeding point with two opposed electrodes on the same handle .

B- Ultrasonic coagulator (Harmonic scalpel) using U/S energy with high frequency to coagulate blood vessels up to 5mm in diameter .

C-Ligation vessel sealing system (Ligasure)): new technology using bipolar electric energy to seal blood vessel up to 7mm in diameter .

D-Laser coagulation : using laser energy to precisely coagulated bleeding points .

4) Surgical clips : using U shaped metal clips which are applied by special instrument were formerly developed for laparoscopic surgery . However , they are now widely used in open surgery .

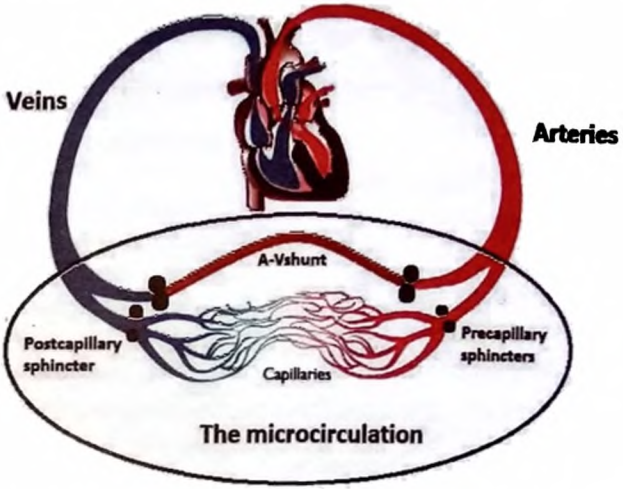
5) Surgical repair of large vessels : important vessels are not ligated and should be repaired with fine suture material using special surgical instrument .

6) Application of certain haemostatic agents :

[a] Gelatin foam

[b] Fibrin glue

Physiology of the circulation



- The circulatory system is composed of **the heart** (the central pump), **arteries** (the feeding vessels), **veins** (the draining vessels) and **blood** (the circulating fluid).
- The function of the circulation is to perfuse the tissues with oxygen and nutrients and carry their waste products to organs of excretion.
- The normal blood volume is about 5-6 liters in adults. Normally, at any moment, 70 % of this amount is present in the veins (capacitance vessels) and heart, 20 % in capillaries and only 10% is in the arteries.
- The capillaries are controlled at both ends by precapillary & postcapillary sphincters. Only 10 % of capillaries are opened at any moment and the other 90% are closed by their sphincters, but they alternate (i e. The opened capillaries will close and another 10 % of capillaries will open). If all capillaries of the body open at the same time. they will suck the whole blood volume as if the patient bleeds into his own circulation
- Also, there are arterio-venous shunts that are normally closed. They open only in certain conditions.

-Under resting condition, the cells get their energy by aerobic (90 %) and anaerobic glycolysis (10 %). Under hypoxic condition, the anaerobic glycolysis accelerates greatly with accumulation of lactic acid & tissue metabolites.

Definition

It is a state of acute circulatory failure with impaired tissue perfusion, disturbed cell metabolism and accumulation of waste products.

Types

I-Hypovolaemic shock

(Hemorrhagic, Secondary or surgical shock)

Aetiology = (Failure of blood.)

1-Blood loss: as in hemorrhage.

2-Plasma loss: as in burn and peritonitis.

3-Water loss: as in diarrhea & dehydration.

Pathophysiology:

I-Compensated stage = reversible stage

*Numerous compensatory mechanisms are performed to:

1) Restore the intravascular volume.

2) Maintain the blood pressure and tissue perfusion.

3) Shift blood from the less vital organs (skin, muscle) to more vital organs (brain & heart).

These mechanisms are:

1- Hypovolaemia & hypotension stimulate carotid sinus and aortic arch baroreceptors leading to

a- increased HR →increased cardiac output → elevation of blood pressure (bl. pr.).

b- Generalized vasoconstriction (VC.) → increased peripheral resistance→elevation of bl. pr.

2- Stimulation of posterior pituitary → secretion of vasopressin (ADH) which causes:

a- Arterial constriction (all over the body except brain & heart) → increased bl. pr.

b- Venular constriction → shift of blood to the arteries.

c- Antiduretic effect → oliguria.

3- Renal ischaemia → secretion of rennin that acts on angiotensinogen producing angiotensin I which is transformed into angiotensin II which induces:

a- arteriolar and venular constriction.

b- Stimulation of the adrenal cortex → secretion of aldosterone that causes Na^+ & H_2O retention.

4-Stimulation of adrenal medulla → secretion of adrenaline & noradrenaline that cause:

a- increased heart rate & contractility → ↑ C.O.P → ↑ Bl. pr.

b- V.C. → ↑ bl. pr.

5-At the level of microcirculation:

a- Catecholamines cause contraction of the precapillary sphincter leading to drop of the capillary pressure → refilling from the interstitial space to replenish the intravascular fluid. This mechanism can refill the circulation by 1 liter /hour in fit patients.

b- If shock is prolonged and the loss is not replaced, decompensation eventually occurs.

II- Decompansated stage (irreversible stage)

* Opening of the A-V.shunts which decreases the tissue perfusion and increases the cell distress that leads to release of histamine & other mediators which cause contraction of postcapillary sphincters. This results in more slowing of the capillary flow.
* Paralysis of the precapillary sphincters due to anoxia & acidosis while postcapillary sphincters are still contracting as they are accustomed to this acidic media. Also the hypoxic capillary damage causes loss of plasma in the tissue & sludging of RBCs in the capillaries with formation of microthrombi.
* Paralysis of the postcapillary sphincter with passage of microthrombi to the circulation → DIC.

-The viscous circle of hypovolaemia → tissue hypoxia → capillary damage → more fluid loss in the tissues → more hypovolaemia is called *"death cycle of Mcdowel"*

Compansated(1) **Decompansated stage (2,3,4)**

Complications

1- Acidosis: due to accumulation of acidic metabolites.
2- Hypothermia:
3- Coagulopathy: due to DIC or dilutional coagulopathy.

} The fatal triad.

4- Electrolytes disturbance: mainly hyperkalaemia due to leak of K^+ out of the cells.

5-Multisystem Organ failure (MOF): renal failure (anuria), heart failure...etc.

Clinical Picture:

I-Mild shock (loss of < 20%of blood volume):

-The non-vital organs (skin, muscle, bone) are affected.

-The patient feels cold with cool, clammy, pale skin.

-Pulse: may be increased.

-Bl. pr: is normal (compensated), but there is postural hypotension.

-Concentrated urine.

II Moderate shock (loss of 20 - 40% of blood volume):

-Liver, kidney, pancreas, spleen are also affected.

-The patient complains of thirst sensation.

-Pulse: tachycardia.

-Bl. pr: hypotension.

-Oliguria (output <0.5ml/kg/hour in adults, <1ml/kg/hour in children &<2ml/kg/hour in infants).

III- Severe shock: (loss of > 40% of bl. volume):

-Brain & heart are also affected.

-The patient is restless (sense of impending death), then becomes drowsy.

-Pulse: rapid weak pulse, and may be arrhythmia that end in cardiac arrest.

-Bl.pr: hypotension.

-Respiration: rapid deep (kaussmaul's) respiration.

Investigations

1-Measuring of the Central Venous Pressure (CVP):

-It can be measured using the usual central venous catheter or better Swan Ganz catheter which can in addition measure the cardiac output (COP) and pulmonary capillary wedged pressure (PCWP). The latter is a good indicator for the left ventricular function.

-Normal CVP. is 5-15 cm water, and not more than 20 cm water.

-The CVP is low in hypovolaemic shock, high in cardiogenic shock & variable in septic shock

- Invasive blood pressure monitoring. It is more efficient in hypotensive states.

2-Blood chemistry:

a) Bl. pH: low b) Serum electrolytes: ↑↑ serum K^+ c) serum lactate ↑↑

d) Bl. gases: ↓ PCO_2 due to hyperventilation and PO_2 is usually normal

Treatment:

I-Resuscitation: (A.B.C.D.E)

A: Airway patency.

B: Breathing normally.

C: Circulation and hemorrhage control by:

- Arrest of bleeding - Restoration of blood volume (Fluid therapy*).

D: Disability: dysfunction of the CNS.

E: Exposure of the patient and seek for hidden injury.

*Fluid therapy: by blood for blood loss, plasma for plasma loss & fluids for fluid loss.

A- Colloids:

1- Blood:

The most effective especially with blood loss.

Disadvantages:

1- It needs time for grouping 2- It may not be available 3- complications of bl. transfusion

2- Plasma:

It supplies albumin, immunoglobulins and when fresh it supplies the coagulation factors.

Disadvantages:

-If collected from many donors. it increases the risk of transmission of diseases.

3- Dextran: (half-life 4- 24h.)

Disadvantages:

a) Haemodilution (not more than 1 liter).

b) Impairs coagulability, and may cause anaphylaxis and nephrotoxioity.

4- Gelatin (Haemagel) (half life 4-5 hours)

- It is most commonly used plasma expander. Reactions are rare.

5- Human albumin (4-4.5 %):

- It is one of the best plasma substitute and reactions are rare, but it is expensive.

6- Hydroxyethyl starches (Volvin)(half life 48 hours) :

- They have long half-life and the lowest risk of anaphylaxis.

7- Synthetic solutions with oxygen carrying capacity: still under trial

B- Crystalloids :

"Three to one' rule - 3 ml of crystalloid should be infused for every 1 ml of blood loss

1) Dextrose 5% (5% glucose) mainly for water replacement.

2) Isotonic saline (NaCl 0.9 %) for sodium loss.

3) Ringer lactate (KCl, NaCl, Ca Cl & Na lactate) for replacement of intestinal fluid loss

Advantages of crystalloids:

a) They are available & cheap.

b) They decrease bl. viscosity, so decrease the after load and increase tissue perfusion.

Disadvantages of crystalloids:

- They escape from the circulation more rapidly.

II- Medications

1) Sedatives.

Morphia is ideal because it:

a) Relives pain & anxiety. b) Increases the cerebral blood flow.

It is contraindicated in head trauma or respiratory depression.

2) Corticosteroids.

They are commonly used to combat stress and they have Na^+ & water retention effect.

3) Vasoactive drugs.

a) Vasoconstrictors: e.g. Noradrenaline. Although they raise blood pressure, yet they:

i)-impair tissue perfusion and aggravate tissue hypoxia.

ii) Increase the after load on the myocardium. So, they should not be used.

b) Vasodilators: e.g. diphenoxybenzamine only after good fluid replacement & improving myocardial contractility. They are given to:

-Improve tissue perfusion -Decrease the after load and heart work.

4) Inotropic drugs: e.g. dopamine, & dobutamine. They are given to improve myocardial contractility and improve renal perfusion (dopamine).

5) Antibiotics: e.g. third generation cephalosporines to avoid septic complications.

NB. All medications should be given IV. not I.M or SC as these two routes are impaired during shock (peripheral VC.) and if shock is corrected, rapid absorption of repeated injections causes toxicity.

III- Other measures

1) Position: elevate the lower limbs about 30 degrees to help venous return and improve flow to other vital organs (e.g. brain). Avoid Trendlenberg's position. as this will increase the heart work to pump blood again to elevated lower limbs.

2) Heating: Cover the patient with ordinary blankets (sense of coolness). Avoid artificial heating as it increases tissue demands of oxygen.

Protocol of management

1-The first step is to correct the preload

Correct the hypovolaemia with IV. fluids, till the CVP returns normal.

-If the cardiac output (measured with Swan Ganz catheter) returns normal, well and good

- If it is still impaired, there is left ventricular dysfunction (confirmed with high PCWP).

2- The second step is to improve the cardiac contractility

Correct heart pumping with inotrops.

-If the cardiac output returns normal, well and good.

- If it is still impaired, this is the time for vasodilators.

3- The third step is to reduce the after load

Reduce the after load with vasodilators.

II- Vasogenic shock

(Failure of the vessels)

Aetiology:

Increased capacity of the vascular bed (due to VD) while the bl. volume remains constant.

Types:

1) Spinal shock: due to

Sympathetic block of the lower half of the body by spinal anesthesia leading to arteriolar & venular dilatation with pooling of blood in the lower half of the body with marked hypotension

2-Neurogenic (primary) shock: due to

1-Sudden fright.

2-Severe pain: e.g.major fracture, blow to the testis or epigastrium.

3-Sight of blood. (Vasovagal shock)

3-Anaphylactic shock: due to

Release of vasoactive substance e.g. histamine or slow reacting substance of anaphylaxis (SRSA) leading to capillary paralysis and pooling of blood in the microcirculation.

Clinical picture

-Sudden collapse with pallor and coldness.

-If it follows vagal stimulation, (parasympathetic), there will be bradycardia and hypotension.

-If it follows anxiety (sympathetic stimulation), there will be tachycardia, sweating & tremors.

-There may be nausea and vomiting.

Treatment

-*Position:* put the patient in supine position with elevation of the lower limbs.

-*Medications:*

1-Vasoconstrictors: have their place here e.g. ephedrine.

2-Atropine: in vasovagal shock to protect the heart from the vagal tone.

3-Corticosteroids & antihistaminic in anaphylactic shock.

4- I.V. fluids: are rarely resorted to.

III- Cardiogenic Shock

(Failure of the pump)

Aetiology:

Myocardial insufficiency leading to decreased cardiac output inspite of good blood volume due to:

1) Massive myocardial infarction.

2) Massive pulmonary embolism.

3) Cardiac tamponade.

4) Severe tacharrythmia or brady-arrhythmia.

5) It may complicate any type of shock, if not properly treated.

Clinical picture:

-Picture of the cause.

-Manifestations of acute heart failure:

*Dyspnea, and may be cyanosis and pulmonary oedema.

*Congested neck veins with high CVP.

*Hypotension with collapse.

Treatment:

- Treatment of the cause e.g. pericardiocentesis in tamponade.
- Inotropic drugs e.g. Dubotamine.
- Intra aortic pulsating balloon may be used in severe cases resistant to inotrops. The balloon is inflated in diastole to help coronary flow and deflated in systole.

IV- Septic Shock

Aetiology:

It is caused by septicaemia especially Gram-ve septicemia.

The endotoxin, which is a component of the cell wall of Gm-ve bacteria is released from dead bacteria and is responsible for all pathological effects in septic shock.

Pathology

The circulating endotoxin causes:

1-paralysis of the capillary sphincters and toxic capillary damage lead to pooling of blood in the microcirculation.

2-direct toxic effect on heart, adrenals & other organs.

3-damage of barrier mechanism of the intestinal mucosa leading to more mobilization of intestinal bacteria & endotoxin to the circulation.

Complications = causes of death

1-Adult respiratory distress syndrome (ARDS).

2-systemic inflammatory response syndrome (SIRS).

3-Multiple organ failure e.g. heart, kidney, liver

4-Acute erosive gastritis

Clinical picture:

The patient passes through two stages:

1-Hyperdynamic (warm) stage:

-Fever (38°C) with warm dry skin.

-Tachycardia & hypotension.

-Normal or high cardiac output (C.O.P).

-The tissues are hypoxic with anaerobic metabolism.

-If not promptly treated, it will pass to the following stage.

2-Hypodynamic (cold) stage:

-Cold clammy skin.

-Tachycardia, severe hypotension.

-Low cardiac output.

-Generalized capillary damage.

-It ends with multiple organ failure, DIC & death.

Treatment: as hypovolaemic shock in addition to:

*Corticosteroids (of doubted value).

*Aggressive broad-spectrum antibiotic therapy.

*Specific monoclonal antibodies to the lipid fraction of the endotoxin.

*Control of the predisposing conditions e.g. DM.

*Treatment of the cause e.g. peritonitis.

Item	Hypovolaemic shock	Vasogenic shock	Cardiogenic shock	Septic shock
Causes	Hypovolaemia	Vasodilatation	Heart failure	Gm-ve septicaemia
Pulse	Usually tachycardia	Usually bradycardia	tachycardia	tachycardia
CVP	Low	Low	High	Variable
Main ttt	IV fluids	Vasoconstrictors	Inotrops	Specific antitoxin
Prognosis	Good	V. good	Bad	V. Bad

Crush Syndrome

(Traumatic anuria - Rhabdomyolysis)

Definition:

It is a renal shutdown following crushing injuries.

Aetiology:

1-Crush (compression) injuries of the limbs.

2-Neglected tourniquet.

Pathology:

Acute tubular necrosis occurs due to:

1-Crushing injuries lead to extravasation of bl. → into muscles → Hypovolaemia.

2-Release of myoglobin from the contused muscle into circulation → precipitates in renal tubules as acid myohaematin → shutdown

Clinical picture:

-*Picture of the cause* e.g. crushed limb (swollen & paralyzed)

-*Oliguric phase:* 2-3 days after trauma.

The urine is small in volume, concentrated and acidic containing acid myohaematin.

-*Polyuric phase:* if the previous phase was treated properly diuresis occurs as the regenerating tubules can't yet concentrate urine .

Treatment:

I-Early cases:

-Correction of shock.

-Remember 3As (antititanic, antibiotics & anti-stress ulcer).

-Flush the kidney with I.V. mannitol.

-Alkalization of urine by I.V. sodium bicarbonate to help excretion of acid myohaematin.

-Fasciotomy to relieve tension over the swollen muscle.

- Splanchnic nerve block may be beneficial.

II Late cases

-Treatment of renal failure e.g. renal dialysis.

-Amputation of the crushed limb.

Cardiac arrest

Definition:

Sudden cessation of effective circulation & respiration.

Aetiology:

1-Hypoxia: The commonest cause due to respiratory obstruction or depression.

2-Hyperkalaemia: It causes arrest in diastole.

3-Heart disease: Myocarditis, cardiomyopathy & arrythmias.

4-Halothane and other drugs: Chloroform, cyclopropane, adrenaline & B blockers.

5-Hypothermia: Spontaneous or induced in open heart surgery.

6-Heavy pulmonary embolism.

Pathology

-Cardiac arrest is a rapidly fatal condition in about 5 minutes as the brain can withstand hypoxia only for 3-4 minutes. Heart, liver & kidneys withstand hypoxia for longer periods.

-Biochemical changes are identical to that of shock e.g. increase in lactate, pyruvates, catecholamines & K^+ in blood with decrease of pH.

Types:

1-Asystole with flat ECG.

The heart is dilated, flappy and cyanosed.

2-Ventricular fibrillation (V.F.)

Very rapid incoordinted myocardial contraction → no pumping.

Clinical picture:

Diagnosis should be done within 30 seconds by:

1- Absent pulse in major vessels as carotid & femoral vesseles.

2-Apnea with or without cyanosis.

3-Unconsciousness.

4-Widely dilated pupil.

N.B. Stethoscope & ECG have no place in diagnosis of cardiac arrest. They waste a very precious time before the life saving cardio-pulmonary resuscitation takes place.

Treatment:-

I-First aid: = External cardiopulmonary resuscitation:

1-Clear airway.

2-Put the patient on flat hard surface like the ground.

3-Mouth to mouth breathing with closure of the nostrils with simultaneous external cardiac massage by compression of the lower 1/3 of the sternum with both hands at a rate of about 60/ min. Breathing / compression ratio should be 2-8

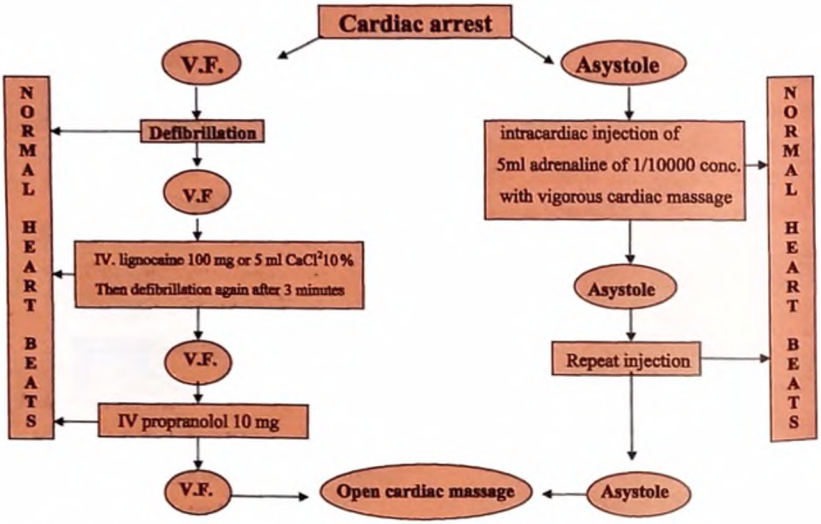
II- At hospital: while maintaing cardiac massage.

1-Enotracheal intubation and artificial ventilation.

2-Venous access usually through cut down.

3-ECG monitoring to detect the type of arrest.

External cardiopulmonary resuscitation



Opens Cardiac Massage.

Indications

1-Persistent asystole or V.F. 2-In open heart surgery.

Procedure

-Lt. 5th intercostal incision (no bleeding occurs as there is no circulation).

-Massage the heart between both hands or by one hand against the back of the sternum

*If it becomes smaller, firmer and pink → hopeful.

*If it remains dilated, flappy and cyanosed → hopeless.

Burn

Definition:

It is coagulative necrosis of tissues by physical or chemical agents.

Aetiology:

I-Physical Agents:

a) Thermal injury: exposure to

-Flame or hot object - hot fluids (scalds).

b) Electricity: electric burn.

c) Irradiation.

II Chemical agents:

Caustics & corrosive (strong acids & alkalies.)

Pathology:

Types:

I- From the medicolegal point of view

1-First degree: *Superficial partial thickness skin burn.* Only part of the epidermis is destroyed. Only erythema develops and it heals very rapidly by regeneration.

First degree burn

2-Second degree: *Deep partial thickness skin burn.* The epidermis and part of the dermis are damaged. It is characterized with bullae formation. Healing can still occur by epithelialization from remnants of hair follicles & sweat glands (epidermal appendages) in the dermis within 3 weeks unless infection occurs.

Second degree burn

3-Third degree: *full thickness skin burn.* The whole epidermis and dermis are destroyed and healing can only occur by migration of epithelium from the margins of the raw area, which is rather slow and associated with scaring and contracture, so grafting is the best treatment.

Third degree burn

4- Fourth stage: Some authors add this stage to refer to burns that involve structures deeper than the skin e.g. muscles and vessels. Complete charring of a limb may occur.

Fourth degree burn

II-From the therapeutic point of view:

1-*Superficial* burn:

- This type includes the first and second degrees in the previous classification.
-At least part of the epidermis remains intact, that may regenerate to epithelialize the raw area
-It is pinkish in colour and hypersensitivite to pin prick.
-Infection may destroy the remaining epithelial islets and transform it into deep one.
-Under favorable conditions, it heals within 3 weeks without need for skin graft.

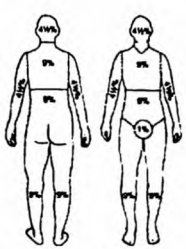
2-*Deep* burn:

- This type includes the third and fourth degrees in the previous classification.
-In which the whole epidermis and epidermal appendages are destroyed, and no hope for spontaneous epithelialization.
-Infection is inevitable.
-It heals with scar formation, unless skin graft is applied.

***Extent:**

-It means the percent of the burnt area in relation to the body surface.
-In adults, it can be calculated by the simple *Wallace's rule of nines.*

*Head and neck→ 9 % of the body surface area.
*Each upper limb → 9% of the body surface area.
*Front of trunk → 18 % of the body surface area.
*Back of trunk →18 % of the body surface area.
*Each lower limb → 18 % of the body surface area.
*Perineum represents 1% of the body surface area.



Rule of nines

N.B. This rule is not applicable for infants & children, as the representative ratios of body parts are different. e.g. the head & neck in a child of 1 year represent about 18% of body surface area.

-Burns are classified according to the extent into:

**Major burns*:

Superficial burns > 15% in adult or > 10% in children & deep burns > 3 %.

**Minor burns*:

Superficial burns < 15% in adult or < 10% in children & deep burns < 3 %.

***Pathological stages:**

1-Stage of wound oedema

-It lasts for the first 3 to 4 days.

-It is characterized by loss of plasma in the tissues (oedema) & to the surface (discharge).

-The plasma loss depends on:

*The extent of the burn: more with wider burn.

*The depth of burn: more in the third degree burns.

-Neurogenic shock, hypovolaemic shock and pulmonary oedema are the leading causes of death in this stage.

2-Stage of wound sepsis (catabolic stage)

-From the 4th day to 30th day.

-Bacterial invasion of the burnt area from:

*bacterial flora. *body orifices (e.g. anus). *environment

-There is toxaemia, constitutional manifestations & Loss of weight due to:

*anorexia (toxic & psychic) * catabolism *sepsis

-Septic shock, pulmonary complications, GIT bleeding (Curling's ulcer) are the leading causes of death in this stage.

3-Stage of wound healing (Anabolic stage)

-After the 30th day.

-The patient is generally better. He regains appetite and gains weight

-The wound heals (if sepsis is controlled).

*Superficial burns heal by epithelialization.

*Deep burns heal by scar formation (unless grafted).

N.B. Superficial burns if managed properly, they can bypass the period of wound sepsis and heal by primary intention (epitelialization).

Complications = causes of death.

I-General complications

1-Shock: neurogenic, hypovolaemic, and septic shock.

2-Chest complications: pneumonia, pul. edema & ARDS.

3-Hepatorenal failure: due to absorption of toxins.

4-Curling's ulcer: usually after the 10^{th} day especially in the 2nd part of the duodenum.

5-Hypoproteinaemia, anaemia, malnutrition and weight loss

II- Local complications

As wounds in general (mention)

Treatment:

I- Minor burns

A-First Aid:

1- Emersion of the burnt area in cold water to:

a) Decrease the noxious effect of heat on tissues.

b) Decrease pain and exudation.

2- Cover the burnt area with sterile towels.

3- Give antibiotic and antitetanic serum (ATS).

B- Definitive ttt

Wash the burnt area with saline and apply non-adherent antiseptic gauze (sofratulle) & sterile dressing (tight, bulky, occlusive),and not disturbed for 2weeks.

II- Major burns:

They are managed according to ATLS in multiple injured patients:

1- Primary Survey: to detect life-threatening conditions.

2- Resuscitation: ABCDE

A) Airway: Ensure patent airway especially in burns of head & neck as inhalation of hot gases may cause serious airway obstruction, which necessitates endo-tracheal intubation.

B) Breathing: Ensure that your patient is breathing well.

C) Circulation: I.V. access (canulation)+ *fluids therapy

D) Dysfunction of CNS:

E) Expose the patient to detect the extent of burn.

3- Secondary survey: To detect other possible injuries.

4- Definitive treatment:

*IV. Fluid therapy:

Aim:

1-Replacement of plasma loss

2-Supplying daily requirement of water as paralytic ileus usually complicates major burn in the first 24-48 h .So oral feeding is avoided in this period.

Amount

*Many formulas were postulated to calculate the amount of fluids therapy. The most popular is *Brooke's formula.* According to this formula:

*In the first 24 hours

Crystalloids (e.g. lactated Ringer 's solution) 1.5 cc **X** % burn **X** BW in kg.

+ Colloids (e.g. plasma or dextran) 0.5cc X % Burn X BW in kg.

The daily requirement of water (about 2000 cc/ day for adult) supplied as glucose5% .

*In the second 24 hours

Half the first 24 hours calculated colloids & crystalloids + the daily requirement of water

N.B. Major burns over 50% are considered 50% to avoid over-hydration (hypervolaemia).

Example: 60 kg man had a 20% burn, Calculate the fluid therapy according to Brooke's formula in the 1st 24 h:
Colloids = 0.5X 20X60= 600 ml plasma
Crystalloids = 1.5 X 20 X 60 = 1800 m Ringer's lactate
So we give 300 ml plasma + 900 ml Ringer's lactate in 1st 8hs then the same amounts along the remaining 16 hours plus 1000 cc Dextrose 5% /12h as body requirement for water.

*We should monitor and check the fluid therapy by

1-pulse & bl. pressure.

2-urine output (at least 0.5ml / kg/h.).

3-CVP. (normal 5-15 cm H_2O).

4-auscultation of the base of the lung.

5-fundus examination to detect papilloedema.

The fluid therapy should be accurately calculated & continuously monitored in the following, risky group.
1- Extremes of age.
2- Patients with cardiovascular or renal disease.
3- Patients with deep burns in the head & neck for fear of brain oedema.
4- Patients with smoke inhalation to avoid pulmonary oedema.

Management of the burnt area

There are three options of management

1-Occlusive dressing (closed) method:

Idea: Recent burns are sterile wounds and the best is to keep it sterile by covering them with sterile occlusive dressing to prevent infection and help epithelialization.

Technique: After cleaning the burn under anesthesia with removing of bullae & debris, the wound is dressed with:

a) Non-adherent gauze (sofra tulle).

b) Bulky absorptive dressing (sterile cotton) to keep the surface dry.

c) Tight occlusive layer (crepe bandage) to prevent oedema, infection & slippage of dressing.

Indications:

1- Burns of the hands & limbs. 2- circumferential burns of the trunk.

-**N.B.** the dressing should be kept undisturbed for 7-10 days unless there is:

1-soaking. 2-marked pain. 3-high fever.

2-Exposure (Open) method:

Idea: If the burn is left exposed to air, the exudates will dry into a crust that covers the surface preventing infection. The epithelium creeps under the crust.

Technique: By nursing the patient under mosquito net with the burn exposed to air, dusted with antibiotic powder every 8 hours.

Indications: (when closed method is not applicable)

1-Burns of the head & neck. 2-perineum.

3-burns of one side of the trunk.

3-Excision & grafting method: -

a) Immediate excision & grafting:

Aim: to excise deep burn & apply skin graft to bypass the phase of wound sepsis

Indication:

-Deep burn not more than 15% of body surface area. Especially in the hand

b) Delayed excision & grafting:

For deep burn treated by exposure method (see before).

***Management of problematic burn**

1-Extensive burn:

a) Autogenous cultures of epithelium on special media (under trial).

b) Homografts (from a relative act as a biological dressing to bypass the period of wound sepsis and may be successfully taken.

c) Meshed autograft: by passing the graft into special set (Mesher) to be fashioned like mesh and can be expanded up to ten times.

Meshed graft

2-Circumferencial burn:

Attention should be paid to the vascularity.

Escharotomy & fasciotomy may be needed.

3-Burn around a joint:

Proper splintage and immobilization in the best functioning position plus physiotherapy.

Special types of burn

1-Electric burn:

Due to effect of electricity on tissue and thrombosis of bl. vessels, sloughing of tissues is wider & deeper than expected in relation to the area of primary injury.

-The treatment is usually by excision & grafting.

2-Chemical burn:

-By chemical agents e.g. corrosives.

-The first aid is removal of soaked clothes and irrigation of the affected area with copious amount of tap water.

-Definitive treatment is usually by excision and grafting if it is deep.

3-Irradiation Burn:

a) Immediate burn: It is due to direct action of irradiation on the tissues in the form of an ulcer covered by thick slough that takes a long time to separate.

-Treatment: conservative dressing until separation of slough. & healing.

b) Delayed burn

-It is due to ischaemic necrosis caused by endarteritis obliterans leading to an enlarging necrotic ulcer (It should be differentiated from local recurrence of the primary malignancy for which radiotherapy was given).

Prognosis:

It depends on many factors:

1-Extent of the burn: the most important factor. Burns > 50% of the body surface area are usually fatal.

2- Depth of the burn .

3-Site of the burn :burns of the head & neck are serious.

4-Age of the patient :bad in extremes of age

5- Pre- existing medical disease e.g. Diabetes, nephritis etc...

Skin Grafts

Definition:

A segment of skin including the epidermis and variable thickness of the dermis transplanted into a raw area not connected to blood supply.

Types

I- According to thickness:

Split thickness graft (thiersch graft)	Full thickness graft (wolf graft)
-composed of the epidermis and part of the dermis.	-composed of the epidermis and full thickness of the dermis.
- Good take (It survives easily).	-Poor take (less chance for survival).
-Donor site heals spontaneously.	-Donor site should be sutured or grafted.
-Bad cosmesis (no colour matching).	-Better sight (better colour matching).
-contracts by time.	-doesn't contract
-less durable (weak resistant to trauma).	-Durable (resists trauma).
-Poor sensation.	-Better sensation.

II-According to source:

1-Autograft: From the patient skin.

2-Homograft: From relative (living human skin).

3-Allograft: From cadaver (dead human skin).

4-Heterograft = xenograft: From animals (pig's skin).

Factors affecting survival of the graft:

1- Good vascularity of the recipient area.

2- Good contact between the graft & the recipient area i.e.

No haematoma or seroma → apply compressive dressing & crepe bandage.

No shearing movement → sutures & splint the part.

3- No infection especially streptococcal pyogens.

Flaps

Definition

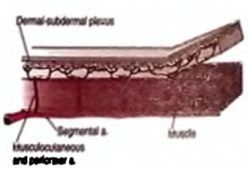
Flaps are tissues transplanted from one site to another maintaining their common blood supply for their nutrition.

Types

According to the pattern of bl. Supply

A. Random pattern flaps:

- They have no anatomically recognized bl. supply.
- They should have length–width ratio not more than 2:1.



Random pattern flap

B- Axial pattern flaps:

- They are supplied by a known artery.
- They have no limitation regarding length to width ratio.
- When axial pattern flap retains only the bl. vessels supplying it without skin attachment they are called **Island flap**.

Axial pattern flap

Island flap

C –Microvascular free flap:

It is an axial flap that is separated completely from its original site then anastomosing the artery & vein of its pedicle to the corresponding vessels in the recipient area.

Microvascular free flap

2) According to their composition

I- Skin flap:

-It is composed of skin, S.C. tissue

-They are classified into

[A]-Random pattern of skin flaps:

1- Local pedicle flaps

-Where the donor area is in close proximity to recipient area

-They include rotational flap, V-Y advancement flap & Z plasty

Lateral abdominal flap

2- Distant pedicle flaps

-Where the donor area is away from the recipient area

-They include:

a) Direct pedicle flaps

-The donor area can be approximated to the recipient area

-They are done in 2 stages

1st stage, the flap covers the raw area but still connected to the donor area by its pedicle

2nd stage, separation of the flap (2-3 weeks later)

- Examples: - lateral abdominal flap
  - cross leg flap & cross finger flap

Cross leg flap

[B] Axial pattern skin flaps

- It is vascularized by a known cutaneous artery.

Groin flap

- It is reliable than random pattern skin flap and have no limitation in length width ratio.

Examples:

1-Groin flap based on the superficial circumflex iliac artery.

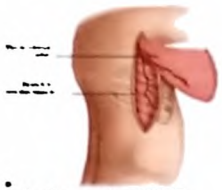
2-Deltopectoral flap based on the 2nd, 3rd, 4th perforating branches of the int. mammary artery

3-Forehead flap based on the superficial temporal artery

II- Fasciocutaneous flaps

-They are composed of skin, S C tissue & fascia

-They are supplied by fasciocutaneous perforators



Latissmus dorsi MCF

II- Myocutaneous flaps

-It is composed of skin, S C., deep fascia & muscle

-It is an axial flap supplied by muscular artery that gives perforating vessels supplying the overlaying skin.

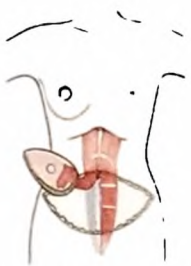
Examples:-

1-Latissmus dorsi MCF based on the thoracodorsal artery from the subscapular artery

2-Pectoralis major MCF based on acromiothoracic artery from the axillary artery

3-Rectus abdominis MCF based on superior epigastric artery from the int. mammary artery.

Pectoralis major MCF



Rectus abdominis MCF

Indications of flaps

1- Large defects that can't be covered by grafts

2- Defect in a weight bearing area e.g. the heal

3- Exposed bone or tendons

4- Exposed artery or vascular anastomosis

5- Defects with large cavity

Multiple (acutely) injured patient

Advanced Trauma Life Support (ATLS)

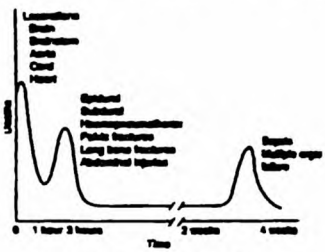
Definition:

Multiple injured patient is that who received severe trauma causing two or more separate injuries, each of them, alone, necessitates hospital admission.

Trauma is the leading cause of death in young adult group and one of the most common causes of death in all age groups world wide.

Death due to trauma occurs in three distinctive peaks:

1-Immediate deaths **(50%)** is within few minutes of trauma. Death during this period results from laceration of the brain, brain stem or spinal cord; injury of the heart, aorta or other big vessels. Very few patients of this category are salvagable. Only preventive measures (e.g. wearing seat belts) can reduce this peak of trauma related deaths.



Peaks of death following trauma

2- ***Early deaths (30%):*** occur within the first few hours after injury (the golden hour(s). Death in this group occurs due to developing airway obstruction, massive internal haemorrhage or CNS compression by collecting bl. So, these deaths are deemed preventable by adequate resuscitation.

3- ***Late deaths (20%)***: occurs days or weeks after trauma mainly due to sepsis or multiple organ failure (MOF). Death in this group is also potentially preventable.

Management:

I- Prehospital care:

It is done by paramedics or experient attendants:

1- Maintain patent airway: by chin lift, supporting the mandible & drawing the tongue forward.

3- Mouth to mouth breathing & external cardiac massage in case of cardiac arrest.

4- Arrest external bleeding by pressure, position (elevation) & packing, rarely, tourniquet is needed.

5- Splinting obvious fractures.

6- During transport, care should be paid not to dislocate a possible unstable fracture spine. The patient is mobilized as one block. Avoid flexion of lumber spines. Cervical spine fracture should be splinted by cervical collar.

II- Hospital care:

A: Priority of patient management (Triage الفرز أو التنقيب)

Evaluation of patients who are most at risk of dying from their injuries and need urgent management from those who can wait. Triage can be done in the following steps:

1- Triage sieve: A rapid survey to separate the dead and the walking patient from the injured.

2- Triage sort: The remaining casulities are classified into four groups

Category I: Critical and can not wait e.g. patients with airway obstruction or catastrophic haemorrhage. Management should be done within few minutes. They take the red colour.

Category II: Urgent. Patients received severe trauma but can wait a short time (30 minutes). They take the green colour.

Category III: Less serious injury. Patients not endangered by delay. Management should be performed after adequate investigations. They take the yellow colour.

Category IV: Expectant i.e. severe multisystem injury with unexpected survival. coloured black.

B–Priority of management:

1-Primary Survey: to diagnose what is killing the patient. It should follow the **ABCDE** rule.

2-Resuscitation: to treat what is killing the patient. It should go simultaneously with the primary survey in the ABCDE rule as follows:

A-Airway:

Airway obstruction is manifested by stridor, tachypnea and working accessory muscles of respiration.

Ensure patent airway by one of the following:

i) Chin lift & drawing the tongue forward.

ii) Jaw thrust: elevate the angle of the mandible.

iii) Clear the oropharynx and suck any secretions.

iv) Oropharyngeal or nasopharyngeal airway.

v) Endortracheal intubation may be needed.

Cricothyroidotomy

vi) Tracheostomy if intubation fails. The best route is through the cricothyroid membrane.

B) *Breathing:* ensure adequate breathing.

Injuries that cause inadequate ventilation and endanger life are:

i) Tension pneumothorax: should be immediately decompressed by insertion of a needle in the 2nd intercostal space in the midclavicular line.

ii) Flail chest: fix the flail segment with plaster.

iii) Open pneumothorax & haemopneumothorax: occlude the wound with dressing and plaster & insert an intercostal tube.

C) ***Circulation:*** ensure adequate circulation.

i) Stop external bleeding by pressure, position & packing .Tourniquet is rarely needed and is associated with many complications. If strongly indicated, apply it and mark the patient with the letter (T) on his forehead or follow him till you remove the tourniquet at the proper time.

ii) Correct shock if present Notice the neck veins:

-If empty (flat), it is hypovolaemic shock. Insert wide bore canula and start fluid therapy with lactate Ringer solution.

-If congested, it is cardiogenic shock due to:

1. Cardiac tamponade.
2. Tension, pneumothorax.
3. Myocardial infraction.
4. Myocardial contusion.
5. Pulmonary air embolism.
6. Hypervolaemia.

D) Dysfunction of CNS

-Level of consciousness: Glasgow coma score (see head injury).

-Signs of laterlalization & cranial nerve palsy.

E) Exposure of the patient:

-It is mandatory for perfect examination and detection of hidden injuries.

***Adjuncts of the primary survey:**

I- Gastric tube and urinary catheter.

II- ECG and pulse oximetry monitoring.

III- Radiological investigations: The only permissible radiological studies are

1- Lateral cervical spines X-ray. 2- AP chest. 3- AP pelvis.

3) Secondary survey:

The secondary survey does not begin untill the primary survey & resuscitative measures (ABCDE) have been completed and the vital functions have been normalized. It includes:

A- History: Ask about **SAMPLE**

- S: Symptom
- A: Allergies.
- M: Medications currently used.
- P: Past illness/ Pregnancy.
- L: Last meal.
- E: Events related to trauma. Tme and type

B- Examination: Meticulous examination from the scalp to the toes.

1-Head & neck:

- Scalp haematoma, depressed fractures.

- Bleeding per mouth, nose or ear.
- Wounds of the neck: risk of air embolism.

3) Thorax:

-Fracture ribs, flail chest, & stove in chest.

- Haemothorax, & pneumothorax.

4) Abdomen:

- Tenderness, ecchymosis, rigidity, & shifting dullness.
- Balance's sign & Keher's sign.

5) Pelvis & perineum:

- Fracture pelvis: pain on distraction of the two iliac spines.
- Perineal tear & haematoma.

6) Spine:

- Fracture, & fracture dislocation: tender spine.
- Spinal cord injury: paraplegia, loss of sphincteric control.

7) Limbs: Test

- Bone: fracture, & dislocation by mobility.
- Vessels: vascular injury by distal pulsation.
- Nerve injury: by motor & sensory loss.

***Adjuncts to the secondary survey:**

A) FAST (Focused Abdominal Ultrasonography for trauma):

Abdominal U/S focused on four areas: the hepatorenal, Lt. Subphrenic, pelvis & epigastrium

B) Additional X rays:

1- X ray abdomen: in erect position to detect air under diaphragm (ruptured viscus).

2- X ray spine & pelvis lateral view to detect fracture & fracture dislocation.

3- X ray limbs: if bone or joint injury is suspected.

C) CT & MRI:

For head injury, spine & pelvic fractures. Also, for questionable chest & abdominal injuries.

d) Diagnostic peritoneal lavage (D.P.L):

In case of abdominal trauma and shock with negative investigations. (see abdominal trauma)

4) Definitive care:

Definitive surgical procedure is performed to repair the injured structures.

Chapter II

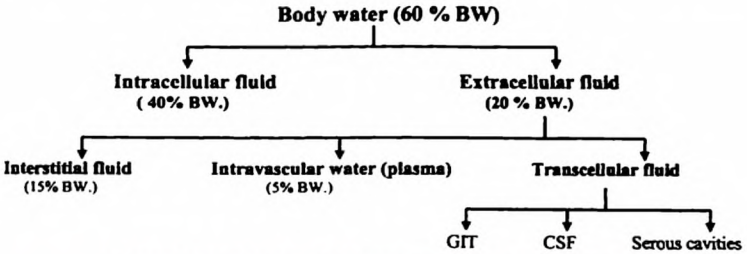
Nutrition in Surgery

Fluids, electrolytes and acid–base Balance

I- Water balance

Normal values

Water represents 60 %, 55% & 75% of the body weight of male, female and newborn respectively.



Normal water balance in sedentary adult in temperate condition

Water Input (ml)		Water output (ml)	
Drink	1500	Urine	1500
Solid food	750	Faeces	100
		Lung	400
Metabolic	250	Skin	500
Total	2500	Total	2500

Water imbalance

I- Water depletion (dehydration)

Aetiology:

I- Decreased intake:

1- Starvation (lack of availability of water).

2- Inability to swallow e.g. in comatosed persons.

3- Restriction of fluids in the postoperative period.

II- Increased loss (not replaced)

1- Excess loss of water from GIT (diarrhea and vomiting) e.g. in intestinal obstruction.

2- Excess loss in urine e.g. in forced diuresis or diabetes insipidius.

3- Excess loss from skin in excessive sweating and fever and evaporation from burns and large wounds.

Pathophysiology:

Loss of pure water results in both hypovolaemia and hyperosmolarity, the later stimulates:

[1] the osmo-receptors or the supraorbital nucleus to secrete ADH that helps to preserve body water.

[2] the thirst area in the hypothalamus that forces the patient to drink excessively

Clinical picture:

1- Intense thirst with excessive weakness. The skin is dry, wrinkled and inelastic.

2- The eyes are sunken and the fontanelles are depressed in infants.

3- Hypotension with rapid pulse and there is marked oliguria with concentrated urine.

4- Nervous manifestations due to nerve cell dehydration (salt poisoning) in the form of excitation, convulsion followed by delirium and coma due to exhaustion of nerve cells.

Investigations:

1- Increased haematocrite value and sodium concentration.

2- High specific gravity of urine with high concentration of nitrogenous waste product.

Treatment:

- The deficit should be first estimated on clinical and laboratory data. Assuming that each 3 mEq elevation of serum sodium concentration above normal indicates a water deficit of about one liter.
- The main line of treatment is fluid replacement with sodium free water e.g. glucose 5%.

2- Water excess (water intoxication)

Aetiology:

[1] Increased intake:

1- Over correction of dehydration with salt free fluids.

2- Excess intravenous infusion of salt free fluids in the postoperative period

3- Colonic washout with plain water instead of saline before colonic surgery.

II- Decreased loss (rare)

Cases of severe oliguria in renal shutdown, ADH treatment or oversecretion.

Pathophysiology:

Water excess leads to increase in the volume of all fluid compartments and causes hypo-osmolarity that inhibits the osmoreceptors and the thirst area in the hypothalamus. Decreased ADH secretion leads to polyuria. Also hypo-osmolarity stimulates aldosterone secretion that leads to sodium retention.

Clinical picture:

1- Headache, drowsiness, nausea & vomiting due to swelling of brain cells. Later convulsions & coma.

2- Increased body weight with pitting oedema.

3- Congested neck veins with high CVP and papilloedema on fundus examination.

Investigations:

1- Low serum sodium concentration (clinical manifestations appear when S. Na^+ is below 120 mEq/l).

2- Low haematocrite value.

Treatment:

1- Restrict water intake.

2- Induction of forced diuresis. In severe cases, dialysis is required if renal failure occurs.

II- Electrolytes balance

-The potassium is the main intra-cellular cation while sodium is the main extracellular cation

-Chloride and bicarbonates are the main extracellular anions while phosphate and sulphates are the main intracellular anions

-Normally the concentration of cations in mEq. must equal the concentration of anions in mEq. to maintain the tissue fluids electrochemically neutral

The chemical composition of plasma and intracellular fluid is

Plasma		Intracellular fluid	
Cations in mEq	Anions in mEq	Cations in mEq	Anions in mEq
Na^+ = 142	Cl^- = 103	K^+ = 150	SO_4^-, PO_4 } = 150
K^+ = 4	HCO_3^- = 27		
Ca^{++} = 5	SO_4, PO_4 } = 3	Mg^{++} = 40	Protein = 40
Mg^{++} = 3	organic acids = 5	Na^+ = 10	HCO_3^- = 10
	Protein = 16		
Total = 154	Total = 154	Total = 200	Total = 200

Sodium balance

Normal Values:

- Normal serum concentration is 135 – 145 mEq /liter.
- Normal dietary requirement is 2 gm. Average dietary intake is 4- 6 gm / day.

Sodium imbalance

1- Sodium depletion (hyponatraemia):

Aetiology:

I- Decreased intake (rare)

1- Restricted intake.

2- Correction of hypovolaemia with salt free fluids or parentral nutrition deficient in Na^+.

II- Increased losses:

1- Increased GIT losses as in vomiting, diarrhea or fistulas (especially high ones).

2- Increased urinary loss as in salt losing diuretics, salt wasting nephritis & adrenocortical insufficiency.

3- Increased loss of the skin, as in excessive sweating or exudation in burns and large wounds.

Pathophysiology:

Hyponatraemia causes excess loss of water in urine to restore the osmotic pressure to normal leading to.

- Polyuria - fall of blood pressure. - Dehydration.

Clinical picture:

1- Muscle cramps.

2- Anorexia, nausea and vomiting.

3- Headache, drowsiness, lassitude and may be fainting.

4- Hypotension, tachycardia, polyuria, dehydration & loss of skin elasticity.

6- Thirst sensation is usually absent due to hypo-osmolarity.

Treatment:

Restoration of the ECF volume by salt containing fluid such as normal saline or Ringer lactate.

2- Sodium excess (Hypernatraemia)

Aetiology:

I- Excess intake:

Excess intake of sodium with food is not usually associated with hypernatraemia as there are many compensatory mechanisms to get rid of the excess sodium. However, excess administration of salt containing fluid e.g. saline in the early postoperative period, when there is a great tendency for sodium retention during stress may result in hypernatraemia.

II- Decreased loss (retention):

1- Hyperaldosteronism (primary or secondary e.g. liver cell failure).

2- Cushing's syndrome.

3- Renal failure

4- Hormones and drugs causing salt retention e.g. oral contraceptives and sex hormones.

Pathophysiology:

Excess sodium causing hyperosmolarity of ECF that retains water with it and drags water from the ICF causing both hypervolaemia and cellular dehydration.

Clinical picture:

1) Puffiness of the face (early) and dependant oedema (late)

2) Thirst sensation.

3) Increased weight.

4) Hypertension.

5) Ascites or generalized anasarca (late).

Treatment:

- Sodium restriction in food.
- Sodium losing diuretics (K^+ preserving diuretic are preferred not to induce hypokalaemia).

Potassium balance

Normal values

Normal serum level is 3.5 –5 mEq/L

Average daily intake is 2-3 gm

Potassium imbalance

1-Potassium depletion (Hypokalaemia)

Aetiology:

I- Low intake:

1- Starvation.

2- Parenteral nutrition with fluids poor in K^+ e.g. maintaining the patients postoperatively on IV fluids that lack potassium e.g. saline and glucose alone.

II- Increased loss

1- GIT loss

a- Repeated vomiting as in gastric outlet obstruction or intestinal obstruction.

b- Severe diarrhea e.g. cholera, ulcerative colitis & villous tumours of the colon and rectum.

c- High fistulas: duodenal, jejunal or ileal fistulas.

2- Urinary loss:

a- Potassium losing diuretics e.g. furosemide.

b- Excess aldosterone or corticosteroids (Conn's and Cushing's syndrome).

III- Intracellular shift of K^+ due to

1) Excess insulin e.g. IV hyper-alimentation or insulinoma.

2) Alkalosis: The potassium shifts inside the cells in exchange for H^+

Pathophysiology:

Hypokalaemia causes

1- Decreased excitability of muscle and nerve tissues leading to muscle weakness & hypotonia.

2- Increased permeability of BBB. to ammonia predisposing to encephalopathy in hepatic patients.

3- decreased the response of renal tubules to ADH leading to polyuria.

Clinical picture:

Many cases are asymptomatic but there may be

1- Nausea, vomiting, abdominal distension and finally paralytic ileus.

2- Apathy, general weakness & slurred speech.

3- Hypotension (hypotonia of the vessel wall).

4- Hypotonia & sluggished reflexes.

5- Arrhythmia, heart block and ECG changes

Treatment:

Potassium administration is very risky as hyperkalaemia causes cardiac arrhythmias and arrest. So at first the definite potassium defect should be precisely calculated using the serum potassium concentration & pH of the blood then the balance is corrected through slow infusion of potassium containing fluids

2- Potassium excess (Hyperkalaemia):

Aetiology:

I- Excess intake.

1) Overcorrecting of hypokalaemia especially if rapid infusion is used.

2) Transfusion of old stored blood as K^+ diffused out from old & haemolysed RBCs.

II- Decreased loss:

1) Renal failure.

2) Addison's disease (hypoaldosteronism).

3) Prolonged use of K^+ sparing diuretics e.g. spironolactone.

III- Extracellular shift of K^+

1) Tissue damage: burn, crushing injuries and haemolysis.

2) Insulin deficiency as in diabetic coma.

3) Acidosis, the K^+ diffuses out of cells in exchange for H^+

Pathophysiology:

Hyperkalaemia induces

- aldosterone secretion that tries to restore the balance through excess K^+ excretion in urine.
- increased excitability of muscle and nerve tissue followed by decreased contractility and conductivity producing manifestations similar to those of hypokalaemia e.g. muscle weakness, ileus.

Clinical picture:

- Numbness and tingling.
- Muscle weakness and malaise.
- Abdominal distension, nausea and vomiting.
- Myocardial depression: bradycardia, arrhythmia and finally arrest in diastole.
- Characteristic ECG changes in the form of wide QRS and hyperacute T wave.

Treatment:

1- Calcium gluconate IV. , as calcium is the pharmacological antagonist of K^+.

2- Alkalinization by sodium bicarbonates as alkalosis induces intracellular shift of K^+.

3- Insulin dextrose infusion as insulin encourages deposition of K^+ with glycogen.

4- If the previous measures fail, dialysis is the final solution.

Calcium balance

Calcium is an important extracellular cation.

Normal values:

Its serum level is 5 mEq/L (9-11 mg %). About 60% of it is ionized and 40% is non-ionized and bound to plasma protein. The ionized part is the active part.

The main hormone regulating the Ca^{++} balance is the parathormone (Ca^{++} raising hormone)

Calcium imbalance

I- Calcium depletion (hypocalcaemia)

Aetiology:

I- Decreased intake.

It is rare except when body needs are increased as in pregnancy also in starvation & dysphagia.

II- Increased loss.

1. Hypoparathyroidism.
2. Vitamin D deficiency
3. Acute pancreatitis
4. Necrotizing fascitis
5. Steatorrhea where the unabsorbed fatty acids combine with Ca^{++} and lost in stool

III- Alkalosis.

Alkalosis decreases the ionized part of calcium.

Pathophysiology:

Hypocalcaemia increases the neuromuscular excitability leading to tetany and arrhythmias.

Clinical picture:

The manifestations depend on the level of serum Ca^{++}. So the disease is divided into the following stages:

I- Latent tetany (S. Ca^{++} is 7-9 mg %)

The manifestations are not apparent and need provocative tests.

1- *Chovestek's sign*: Tapping over any superficial nerve causes contraction of the related muscles such:

- Tapping over the facial nerve in front of the ear causing contraction of the facial muscles.
- Tapping over the peroneal nerve over the head of fibula causing contraction of the peroneal muscles.

2- *Trousseau's sign*: Applying the sphygmomanometer cuff over the upper arm and raising the pressure over the systolic pressure for 2-5 minutes induces carpal spasm.

3- *Erb's sign:* Low amperage electric stimulation (< 4 mili-ampers) can cause muscle contraction (Normally not less than 8 milliampers)

II- Manifest tetany (S. Ca^{++} is below 7 mg %)

1- Irritability, restlessness, tingling and numbness around the mouth and fingers.

2- Muscle twitches, carpopedal spasm and may be convulsion.

3- Spasm of facial muscle may cause risus sardonicus and prevertebral muscle may cause opisthotonus.

Treatment:

- Vitamin D2 (calciferole) or better Dihydrotachysterole (AT 10) that is more potent than calciferole.
- Supplementary oral calcium and may be magnesium.
- Correction of alkalosis if present.
- Attacks of tetany can be ended with IV. calcium gluconate slowly.

2- Calcium excess (Hypercalcaemia)

See hyperparathyroidism

III-Acid- Base Balance

Normal blood pH of 7.4 ± 0.04. Maintaining the pH of body fluids within a narrow range is essential for

1- Optimal function of body enzymes.

2- Proper distribution of electrolytes.

3- Ideal function of myocardium.

4- Normal vascular resistance e.g. alkalosis causes cerebral and coronary vasoconstriction.

Regulation of acid-base balance

- About 15000 mEq of H^+ is produced by normal metabolism per day yet the pH of the extracellular fluid is maintained within its narrow normal range by the aid of body buffers. which include

A- Chemical buffers

Chemical buffers are solutions that resists the changes in its pH when an acid or a base is added to it

1- *Protein buffer*:-including haemoglobin – proteins can behave as a weak acid or weak base

2- *Bicarbonate buffer*: one of the most important buffer in the body. When it buffers strong acid, volatile H_2CO_3 acid is formed that can be excreted through the lung as CO_2

3-*Phosphoric acid buffer*: (NaH_2PO_4 /Na_2HPO_4) also one of the important buffers.

B-Respiratory buffering system

Lung can participate actively in acid-base balances through its excretion or less effectively retention of CO_2. The hyperventilation during acidosis is one of the most effective buffering mechanisms.

C- Kidney buffering

Kidneys share in regulation of acid/base balance through three main actions:

1- Active reabsorption of filtered H_2CO_3.

2- Excretion of titerable acids e.g. (Na_2HPO_4, NaH_2PO_4, H^+).

3- Excretion of ammonia salts (NH_3 - NH_4).

*Chemical buffers represent the first line defense against acid/base disturbance, absorbing the immediate shock of addition of strong acid or base to the body blood. Lungs represent the second line of defense, acting mainly against acidosis. The third line of defense is the kidney through excretion of acid or base.

Acid base imbalance

Respiratory acidosis

Definition:

Acidosis due to retention of excessive amount of CO_2 secondary to hypoventilation.

Aetiology:

1- Respiratory center depression, due to anaesthesia, barbiturates, opiates or head trauma.

2- Neuromuscular disease, deficient respiratory movement of the chest wall due to chest deformity, myasthenia gravis, poliomyelitis, chest trauma, neuromuscular depression by drugs.

3- Pulmonary diseases: COPD, pulmonary fibrosis, pulmonary edema, extensive pneumonia.

Pathophysiology:

A) Compensation:

1- Chemical buffers: Excess formation of bicarbonate through hydration of CO_2.

2- Respiratory buffer: The high PCO_2 (hypercapnia) stimulates the respiratory center through the carotid bodies & the medullary chemoreceptors leading to increased depth and rate of respiration

3- Kidney buffer: It is the most important in respiratory acidosis. It acts through:

i- Excretion of titrable acids.

ii- Increased reabsorption of bicarbonate

iii- Excretion of ammonium chloride resulting in hypochloraemia

B) Decompensation:

With time the pH of CSF is restored to normal, due to active transport of bicarbonate into CSF so removing the main stimulus for respiration.

Complications:

1- Cardiac arrythmia due to stimulatory of CO_2 on cardiac muscle.

2- CO_2 narcosis, delirium and coma due to action of CO_2 on CNS.

3- Hyperkalaemia due to compensatory K^+ out diffusion of cells in exchange of H^+.

4- Manifestations of hypercalcaemia due to increase of ionized Ca^{++} by the acidic plasma.

Clinical picture:

1. Clinical picture of the cause.
2. Respiration: early Kussmaul's respiration but later respiratory failure with cyanosis.
3. Irritability, hallucination, delirium and coma
4. Cardiac arrythmia in the from of tachyarrythmia
5. Manifestation of complications

Investigations:

1. Blood pH is low or low normal.
2. Blood HCO_3 is high and PCO_2 is high (> 44mmHg).
3. Serum K^+ is high, while serum Cl^- is low.

Treatment:

1- Artificial mechanical ventilation to correct hypoventilation. However, great care should be taken not to correct hypoxia totally as this may diminish the sensitivity of respiratory center to hypercapnia, the fact that deteriorates respiratory failure and acidosis. So O_2 should be given in low concentration and under supervision.

2- Treatment of the cause e.g. respiratory infection with antibiotic & bronchodilators.

3- Treatment of complications.

Metabolic acidosis:

Definition:

Acidosis due to accumulation of metabolic acids or excess loss of bases leading to depletion of H_2Co_3.

Aetiology:

I- Addition or retention of excess amount of acids:

1- Diabetic Ketoacidosis, due to excessive fat metabolism in diabetes mellitus with production of large amount of ketoacids (ketone bodies) e.g. acetoacetic acid.

2- Lactic acidosis, due to over production of lactic and pyruvic acids from anaerobic tissue metabolism in shock &sepsis.

3- Renal failure: the failed kidney cannot excrete the normal dietary acid load.

4- Uretero-sigmoidostomy due to active absorption of H^+ & Cl from urine by the colon.

II-Base loss:

1. Loss of pancreatic or intestinal secretion as in:

   i) Pancreatic fistula. ii) Severe diarrhea. iii) Intestinal fistula.

2. Addison's disease (hypocorticism): Lack of aldosterone hormone leads to loss of H_2CO_3 in urine.

Pathophysiology:

A) Compensation:

1-Chemical buffers, especially bicarbonates and phosphoric acid buffers:

$NaHCO_3 + HCl \longrightarrow NaCl + H_2CO_3$

$Na_2HPO_4 + HCl \longrightarrow Na\,Cl + NaH_2PO_4$

The resultant H_2CO_3 is excreted by the lung as CO_2 and NaH_2PO_4 is excreted by the kidney.

2-Respiratory buffers: Stimulated respiration (Kussmaul's respiration) leads to get rid of excess CO_2

3-Kidney buffers: Within 48 hours kidneys compensate for acidosis as before

B) Decompansation:

If the acid load is big or if one or more of these compansatory mechanisms are impaired as in renal failure or other renal causes of acidosis the pH of blood drops with resultant fatal acidaemia.

Complications and clinical picture:

As in respiratory acidosis

Investigations:

1-Blood PH is low or low normal.

2-Blood H_2CO_3 is low and PCO_2 is normal or low.

4-S. Cl^- is high or normal, and S. K^+ is usually high.

Treatment:

I-Mild & moderate acidosis: treatment is usually towards the cause e.g. correction of hypoxia, improving tissue perfusion, treatment of diabetic ketoacidosis.

II-Severe acidosis:

Sodium bicarbonate is given according to the following formula:

- $NaHCO_3$ (needed) = body weight × base deficit × 0.2

(Where: Base deficit = plasma bicarbonate difference and 0.2 = the volume of extracellular fluid as a fraction of body weight.)

-Half of this calculated amount is given intravenous, and then the other half is given slowly under repeated monitoring of blood pH and plasma bicarbonate concentration.

-In diabetic ketoacidosis, bicarbonates are rarely needed as insulin therapy leads to rapid metabolism of ketone bodies to bicarbonate.

Respiratory alkalosis

Definition:

Alkalosis due to excess wash of CO_2 secondary to hyperventilation.

Aetiology:

Hyperventilation may be due to:

1-Hypoxia: as in high altitude, it stimulates the respiratory center through the carotid bodies.

2-Hyperpyrexia: Fever causes hyperventilation to get rid of excess heat.

3- Hysteria and psychosis:

4-Infection: sepsis especially meningitis.

5-Iatrogenic: e.g. hyperventilation under general anesthesia.

6- Induced: e.g. self-induced hyperventilation.

Pathophysiology:

A) Compensation:

1- Chemical buffers: protein and phosphoric acid buffers release their H^+ ion to correct alkalosis.

2-Respiratory buffer: The hypocapnia and elevation of plasma pH tend to suppress respiratory center activity. This tends to brake the hyper ventilation.

3-Kidney buffer: Renal buffering mechanism acts through:

a)Reduction in H^+ and titerable acids excretion.

b)Decrease absorption of HCO_3 leading to its loss in urine

These compensatory mechanisms are associated with retention of Cl & K^+ diuresis thus hyperchloraemia & hypokalaemia are usually associated problems.

B) Decompensation:

If the buffering mechanisms are deficient the pH of blood rises with resultant fatal alkalaemia.

Complications:

1) Cardiac muscle depression and arrhythmia.

2) Delirium and disorientation.

3) Tetany due to decreased ionized Ca++ by plasma alkalinity.

4) Hypokalaemia due to intracellular shift of K^+ in exchange of H^+

Clinical picture:

1- Clinical picture of cause e.g. hyperventilation.

2- Nervous manifestations: dizziness with tingling sensation in the extremities and around the mouth. These manifestations are due to cerebral vasoconstriction secondary to hypocapnia.

3- Tetany in the form of carpo-pedal spasm with hyperexcitability and numbness of the fingers.

4- Manifestations of complications e.g. hypokalaemia, arrhythmia.

Investigations:

1-Bl. pH is elevated or normal.

2-blood H_2CO_3 is normal or decreased.

3-PCO_2 is decreased.

4-Serum K^+ is usually low.

5-Serum chloride is usually high.

Treatment:

1- Treatment of the cause of hyperventilation.

2-Alkalosis under anaesthesia can be corrected by rebreathing of the expired air while in hysterical cases, breathing in closed bag usually corrects the imbalance, if failed, good sedation is needed.

Metabolic alkalosis

Definition:

Alkalosis due to excess loss of acids or addition of excess bases leading to retention of H_2Co_3

Aetiology:

I-Excessive loss of acids as in:

1) Repeated vomiting or prolonged nasogastric suction that lead to loss of hydrochloric acid as occurs in gastric outlet obstruction and Zollinger Ellisson's syndrome.

2) Loss of H^+ in urine as in:

(a) Conn's syndrome (primary hyperaldosteronism) & Cushing's or diuretic therapy.

(b) Bartter's syndrome (hyperplasia of juxtaglomerular apparatus resulting in high rennin-Angiotensin-aldosterone concentration) leading to loss of H^+ in urine.

3) Loss of H^+ intracellular, this occurs in hypokalaemia as H+ shift inside the cells in exchange of K^+

II-Addition or retention of bases

1) Milk alkali syndrome, due to compulsive drinking of milk & antacids in treatment of peptic ulcers.

2) Excess H_2CO_3 administration: e.g. over-correction of metabolic acidosis.

3) High renal threshold of excretion of bicarbonates

4) Volume contraction of the extra cellular space leads to relative increase in H_2CO3 concentration.

Pathophysiology:

A) Compensation:

1) Chemical buffers: especially protein and phosphate liberate H^+ ions. Also movement of H^+ out from the cells in exchange for K^+ tends to correct the imbalance.
2) Respiratory buffering: the plasma pH suppresses the respiratory system so leading to hypoventilation and CO_2 retention. However the resulting hypoxia tends to release the respiratory center from this suppression.
3) Renal buffers: Increased renal excretion of H_2CO_3 and decrease renal tubular excretion of H^+ with production of alkaline urine.

B) Decompansation:

-If these physiological buffers are inefficient, the plasma bicarbonates accumulate in the extra-vascular fluid with fall of blood pH and resultant alkalaemia.

Complications & clinical picture:

Similar to respiratory alkalosis with the exception of hyperventilation of respiratory alkalosis instead there is compensatory hypoventilation.

Investigations:

1- Blood pH is high or normal.
2- Plasma H_2CO_3 is high and PCO_2 is normal or high.
4- Serum K^+ is usually low and serum chloride is usually low.

Treatment:

1)Treatment of the cause is the mainstay of the treatment e.g. pyloric obstruction & Conn's syndrome.
2) Correction of hypochloraemia and hypokalaemia by potassium chloride infusion also help correction of the alkalosis.
3) In cases of pyloric stenosis, simple saline infusion is very beneficial because the kidney will complete the task with excess bicarbonate and chloride retention along with sodium diuresis.
4) In more severe case, ammonium chloride infusion or 0.1 N hypochloric acid.

A/B imbalance	*Causes*	PCO_2	HCO_3	K^+	Cl^-
1-Respiratory acidosis	Retention of CO_2	High CO_2	Normal or *high*	↑	Usually low
2-Metabolic acidosis	Excess acids or base loss	Low or normal	Low	↑	Usually high
3-Respiratory alkalosis	Washout of CO_2	Low	*Usually* low	↓	Usually high
4-Metabolic alkalosis	Loss of acid or adding bases	Normal or high	Usually high	↓	Usually low

Malnutrition in surgery

Most of the patients who are going to have surgical operations are fairly nourished so they can withstand the stress of surgery and starvation for few days following surgery. Others are either preoperatively malnourished or become so post-operatively due to complications or delay in resumption of normal diet. Those patients require nutritional support.

Causes of malnutrition in surgical patients

1) Starvation

1- Social causes as poverty, senility and ignorance.

2- Anorexia is caused by malignancy and sepsis

3- Inability to eat due to malignancy of the oral cavity, pharynx and oesophagus

4- Repeated vomiting due to gastrointestinal diseases and malignancy.

5- Maldigestion and malabsorption , due to inflammatory bowel diseases, short bowel syndrome, high intestinal fistula, blind loop syndrome

6- Restricted oral intake due to postoperative paralytic ileus, surgical intestinal anastomosis

2) Hypercatabolism, due to :-

1-Major trauma accidental or surgical

2-Major sepsis e.g peritonitis

3-Severe acute pancreatitis.

4-Chemotherapy or radiotherapy

The drawbacks of malnutrition on surgery

Malnutrition increases postoperative morbidity and mortality

1-Impaired wound healing: dehesence of abdominal incision and intestinal anastomosis leading to burst abdomen and intestinal leak.

2-Suppressed immunity and increased susceptibility to infection

3-Mental depression and physical exhaustion that delay recovery and increases hospital stay

Diagnosis

Malnutrition is diagnosed by many tests the simplest parameters are:-

[1] Anthropometric parameters

1- Recent weight loss of 10% or more

2- Body weight is less than 80% of the ideal for weight

3- Mid arm circumference is less than 80% of the ideal value (indicative of protein loss)

4- Triceps skin fold thickness (indicative of fat loss)

[2] Laboratory parameters

1-Serum albumin less than 3.5 g % is indicative of severe protein loss

2-Total lymphocytic count below 1.2x10

Nutritional support of surgical patients

All patients with malnutrition or those at risk of malnutrition need nutritional support.

[1] Oral nutrition

Undoubetly, it is the easiest, safest, most pleasant, most physiological and efficient method of nutrition. Liquidized food may be used if mastication or deglutition is weak

[2] Entral nutrition (tube feeding)

It is indicated when oral intake is not possible but with functioning small bowel such as:-

1- Comatosed and seriously ill patient

2- Patient with mouth, pharynx or oesophago-gastric lesions

Types:-

(1) Nasogastric tube feeding

Liquidizing food, clinifeed or supplements are given through a nasogastric (Ryle) tube

(2) Gastrostomy or jejunostomy feeding. Jejunostomy formulas are driped slowly in to GIT

Complications:

A-Complications of the tube: Slipping, blockage, infection or bleeding at the site of insertion

B- Other complications: Reflux, vomiting, diarrhea, aspiration, pneumonia, hyperglycaemia

[3] Parentral nutrition (intravenous nutrition)

When GIT is out of function, nutrients are given intravenously either through:

a) Peripheral line, through a peripheral vein

b) Central line through large vein with high rate of flow. It is administrated through a central venous catheter (CVC) which is percutaneously inserted into the internal jugular or subclavian veins threaded to the superior vena cava. The high flow of blood dilutes hypertonic solutions to avoid thrombophlibitis.

Total parentral nutrition

When entral feeding is not possible, total parentral (intravenous) nutrition is indicated

Indications:

1- Comatosed and seriously ill patients who cannot tolerate oral and entral feeding, e.g they regurgitate and aspirate food on tube feeding.

2- Patients where GIT is out of function or inadequate for nutrition e.g short bowel syndrome, high intestinal fistula, paralytic ileus

3- Infants with congenital anomalies, enzyme deficiency or malabsorption

4- Patients with hypermetabolic state e.g major trauma, major burn, major postoperative complications or high output fistulas

Components of TPN

A balanced solution for TPN should contain :-

1- Water: at least 3 litres per day for adult

2- Calories: combined carbohydrates and fat. Adult patients need about 2000 k cal / day but those with hypermetabolic state may need up to 4000 k cal / day

3- Protein: supplied as amino acids. Normal adult individuals need about 70 gm of protein but hypercatabolic patient may need 150 gm of protein (30 gm nitrogen)/day

4- Electrolytes e.g Na, K, calcium, phosphate & magnesium

5- Vitamins and trace elements

Now a balanced mixed solution of 3 litres is commercially available or prepared in the hospital pharmacy containing all nutrients required for 24 hours period which can be taken at home especially in ambulatory patients on long term TPN e.g short bowel patients (home TPN). The solution can be given overnight and the patient can go work by day or given through the 24 hours

Monitoring TPN patients:-

1- Blood glucose should be monitored every 6 hours

2- U & Es should be checked daily

3- Calcium, phosphate should be checked twice weekly

4- If fever develops, blood culture should be done and if sepsis can't be attributed to any other cause CVC should removed and its tip is sent for culture & sensitivity

- The site of catheter insertion should be aseptically dressed twice weekly

Complications

[1] Complications related to the catheter:-

1- Pneumo or haemopneumothorax and arterial puncture may complicate catheter insertion

2- If catheter is not probably positioned, it may cause arrhythmia or venous wall erosion and haemopericardium

3- Thrombosis or thrombophlebitis of the big veins

4- Septicaemia due to septic thrombus around the catheter

5- Air embolism: as the pressure in neck veins may be negative during inspiration. So we should keep the CVC always closed system

[2] Metabolic complications

1- Fluid overload

2- Hyperglycaemia or hypoglycaemia

3- Hyperosmolar dehydration

4- Electrolyte disturbance

5- Hepatic cholestasis and later cirrhosis

Chapter III

Infection

Definitions:

- Contamination is just innoculation of the organism into the tissues
- Infection is invasion of body tissues by pathogenic organisms causing pathological changes.
- Sepsis is systemic manifestations of infection.

Aetiology:

- Infection results when microorganim virulence overcome host resistance.

Predisposing factors:

a) Virulence of the organism : exotoxins ,endotoxins , ... etc

b) Size of inoculum : > 100,000 organisms/1 gram tissues.

c) Presence of nutrient source for the organism : hematoma or necrotic tissues.

d) Breakdown in the host's defense: immunodefiency,diabetes,cancer ..... etc

Pathology

**Local phase of infection:*

- After bacterial invasion , inflammatory response can abort infection
- If the extent of tissue injury and number of bacteria exceed the capacity of the host to terminate an infection locally, infection is established.

**Systemic phase of infection: sepsis:*

- Failure of local control leads to bacterial invasion of blood (bactremia and septicemia.)
- Release of bacterial exo and endotoxins lead to sepsis , septic shock multiple organs failure (MOF) and death

Clinical picture:

I- Clinical picture of local infection: signs of inflammation ie. (pain), rubor (redness), calor (hotness) , tumor (swelling) & functio laesa (loss of function).

II- Clinical picture of sepsis :

- Body temperature <360C or >380C
- Heart rate >90 beats/min
- Arterial hypotension: SBP* <90 mmHg; MAP* <65 mmHg
- Respiratory rate >20 breaths/min or PCO2* <4.3 kPa*
- White cell count <4000 or >12000/mm^3
- Coagulation abnormalities: INR* >1.5 or APTT* >60 seconds
- Thrombocytopenia, hyperbilirubinemia & hypercalcemia

***SBP**, systemic blood pressure , **MAP**, mean arterial pressure: **PCO2**, carbon diaxide partial pressure*
KPa, kilopascal INR, international normalized ratio ***APTT***, activated partial thromboplastin time*

I. Specific infections

When the pathological changes occuring in the tissues are specific to the organism

1- Tetanus

Definition:

It is a specific type of wound infection with serious neurological sequel and high mortality.

Aetiology:

-Causative organism:

Clostridium tetani: motile noncapsulated Gm +ve bacilli with terminal spore *(drumstick)*. It is obligatory anaerobe & exotoxin producer.

Drumstick spores

- Routes of infection:

1-Direct contamination of wounds by manured soil.

2-Infection of surgical wounds by poorly sterilized catgut (*postoperative tetanus*).

3-Infection of the genital tract of the mother (puerperal tetanus) or the umbilical stump of the baby (*tetanus neonatorum*).

- Predisposing factors :

1- Deep punctured wounds.

2- Hypoxic wounds due to tourniquet, tight bandage, hypoxia or shock.

3- Associated pyogenic infection consumes oxygen and produces anaerobic media for tetanus.

4- Wounds containing foreign bodies, blood clots & necrotic tissues .

Pathology:

-The organism secretes a powerful neurotoxin that reaches the CNS either:

a) directly from the wound along neuronal axons & perineural lymphatics .

b) through the bl. stream to motor end plates allover the body then along their axons to the CNS.

-After the exotoxin reaches the CNS it becomes fixed to anterior horn cells (AHCs) & motor nuclei of cranial nerves in brain stem. (the fixed neruotoxin can't be neutralized by antitoxin)

-The exotoxin has two noxious effects :

*Local action on motor end plates by inhibition of choline estrase enz., so, acetyl choline accumulates in excess amount.

*Central action on AHCs causing hyperexcitability & convulsion in response to minor stimuli.

Complications & causes of death:

1- Muscle rupture & bone fractures.

2. Heart Failure.

3- Asphyxia.

4- Hyper pyrexia.

Incidence:

It is common in wars and traffic accidents.

Clinical picture:

I- Incubation period: 1-15 days (It may be months in immunized persons).

II-Prodromal symptoms:

-Irritability, mild fever, tachycardia, insomnia & headache.

-The wound appears inflamed with the surrounding muscles twitching.

III- Stage of tonic contraction:

(The 1st muscles affected are the muscles of the head & neck due to their short axons).

- Trismus (lockjaw) & dysphagia.
- Facial muscles spasm → *Risus sardonicus* = bitter smile.
- Stiffness of the neck.
- The rigidity then spreads gradually to the trunk.
- The peripheral muscles of the limbs are usually spared.

Bitter smile

IV- Stage of clonic contraction:

- Convulsive attacks at first are stimulated by noise, light, air & movement. Later, they occur spontaneously .
- During convulsion, the body is arched usually backward (opisthotonus), forwards (emprosthotonus) or to one side (pleurosotonus) during attacks .
- Between the attacks, the tonic spasm persists.

opisthotonus

- Later, hyperpyrexia , & serious asphyxia ensues. The patient remains conscious till death.

Clinical types of tetanus:

I- According to the mode of transimission:

1- Postoperative tetanus.

2- Purperal tetanus & neonatal tetanus.

3- Cryptogenic tetanus: a mild form without apparent wound.

4- Latent tetanus: occurs after reopening of old wounds containing dormant spores.

II- According to the site of affection:

1- Cephalic tetanus: due to wounds of the head & neck .It has a shorter incubation period. It affects mainly the muscles of the head and has a bad prognosis.

2- Splanchnic tetanus: It affects mainly muscles of deglutition & respiration.

3- Local tetanus: occurs in immunized individuals. Only the surrounding muscles are affected.

III- According to the course of the disease:

1- Acute tetanus: It occurs in non immunized persons. The period between the trismus and the onset of convulsion is short and the disease is usually fatal.

2- Chronic tetanus: long incubation period, mild symptoms & favorable prognosis.

Investigations:

Polymorphnuclear leucocytosis may be present.

Differential diagnosis:

1-Other causes of trismus: e.g. parotid abscess, dental infection, & quinsy.

2-Strychnine poisoning: - Convulsions affect mainly the extremities.

- The muscles are completely relaxed between convulsion.

3-Tetany: - Hands & feet are mainly affected (carpopedal spasm).

- +ve Chvostek & Troussau signs. Serum Ca^{++} is low.

4-Meningitis: - Neck rigidity starts first .

- CSF exam. is diagnostic .

5-Rabies: - history of animal bite.

- Convulsions occur on drinking leading to hydrophobia.

- Muscles of deglutition & respiration are mainly affected.

Treatment:

I- Prophylaxis:

A- Proper surgical procedures:

-Adequate wound debridment, removal of foreign bodies & necrotic tissues.

-Avoid tight bandage and tourniquet.

B- Prophylactic immunization :

1- Active immunization: by tetanus toxoid .Routine immunization in infancy by 3 injections of 1ml. at 2 months interval with booster does every 5-10 years & booster dose at time of injury.

2-Passive immunization: by antitetanic serum (ATS) or better by human antitetanic globulin (ATG) (to avoid serum sickness & anaphylaxis) 1500: 3000 IU. ATS. IM. (after sensitivity test) or 250 U. ATG. are given together with toxoid immunization at time of injury.

II- Treatment of established cases:

A-Antitoxin: in large dose, 200.000 IU ATS. or better 3000 U ATG. are given IM to neutralize the free (unfixed) exotoxin. 50.000 IU. ATS. is given after one week .

B- Antibiotic: in large dose ,10-40 million IU penicillin G/day are given in divided doses.

C- Control of convulsion:

In mild cases, convulsions are controlled by barbiturates or promazine or both.

In severe cases, curare or flaxedyl (muscle relaxant) with artificial ventilation is necessary

D- Control of hyperthermia .

1-Cold fomentations 2- antipyretics 3-largactil

E- Nursing care : in ICU.

- The patient is put in dark, quiet, air conditioned room.
- Patient is cared as comatosed one (see head injury)

F- Wound care: (only after antitoxin & antibiotic)

- Drainage of the wound & excision of dead tissues.
- The wound should be laid open and irrigated with H_2O_2

Prognosis :

Mortality rate is 30 -60 %

2-Gas Gangrene
(Clostridial myositis)

Definition:

It is acute clostridial myositis associated with rapidly spreading gangrene usually with gas formation and severe toxaemia.

Aetiology:

A-Causative organisms : They are of the clostridium group that fall into two groups.

A- Saccharolytic group: Cl. Welchii, Cl. septicum. Cl. oedematiens .

B- Proteolytic group: Cl. sporogenes, Cl. histolyticum & Cl. tertium.

They are gram +ve bacilli, anaerobic & spore forming.

II-Route of infection:

Direct contamination of wounds by organisms usually from clothes contaminated by faecal matter. Less commonly, contamination may be by soil & manure.

III-Predisposing factors:

As tetanus.

Pathology:

- Presence of dead or ischaemic muscle is essential for the evolution of the disease.
-The saccharolytic group begins the story with their toxins that cause fermentation of muscle glycogen and liberation of CO_2 & H_2.These gases dissect the tissue planes infront of infection & cause thrombosis of vessels & haemolysis of RBCs.

-The proteolytic group will complete the story by attacking the dead muscle splitting their protein into ammonia & H_2S, which are responsible for the characteristic musty odour of the wound. Also, H_2S combines with iron liberated from haemolysed RBCs. forming iron sulfide that gives the characteristic greenish black colour of the dead muscle.

Complications & causes of death:

1- Severe toxaemia & septicaemia.

2- Haemolytic anaemia & jaundice.

3- Multiple organs failure: liver, kidney, & adrenals.

Incidence:

It is common in war injuries rather than civilian accidents.

Gas gangrene

Clinical picture

- Incubation period: 1-2 days.

- Prodromal symptoms:

The patient appears pale, irritable with mild fever.

The patient complains of pain & numbness in the wound.

- Full blown picture:

General:

–Toxaemia, pallor & fever (subnormal temperature in severe cases).

–Hypotension, vomiting & jaundice.

Local:

-The wound appears swollen with tense sutures and the skin stains greenish yellow.

-Highly offensive (musty odour) brownish discharge comes out of the wound.

-The muscles aren't viable (dark, flabby & doesn't contract on grasping by a toothed forceps or bleed on cutting). They change their colour to dusky red then greenish black.

-Crepitus under the skin due to gas formation.

Investigations:

1- Swab from the depth of the wound → direct exam. → Gram +ve bacilli.

2- Plain X ray of the affected limb → Gas under the skin.

Differential diagnosis:

Not all gas containing wounds are clostridial infections.

Treatment:

I-Prophylactic ttt:

1- Correction of shock & hypovolaemia.

2- Proper surgical technique:

- Adequate debridment and removal of foreign bodies.
- Adequate debridment of tissues till reaching the healthy muscle fibres (red, firm contract on pinching and bleed on cutting)
- Avoid tourniquet , tight pack or bandage .

3- Prophylactic antibiotics e.g. penicillin.

4- Prophylactic antiserums : polyvalent anti gas gangrene serum IM injection (obsolete).

II Treatment of established cases:

1- Correction of shock: Blood or plasma transfusion.

2- Correction of local hypoxia by

-Hyperbaric O_2 therapy under O_2 tent (suppress toxin production by the organism)

- Irrigation of the wound with hydrogen peroxide.

3- Antibiotics: high doses of crystalline penicillin 10-40 million unit in divided doses.

4- Wound debridment: in localized form, adequate muscle debridment down to healthy tissues.

5- Amputation: in massive type high amputation is done as a life saving procedure.

Prognosis:

Mortality rate 20 – 50 %

3- Erysipelas (الخَمْرَة)

Definition:

Highly infectious, rapidly spreading, non-suppurative streptococcal lymphangitis of the skin.

Aetiology:

I-Causative organism: Streptococcus erysipelatis (haemolytic streptococci).

II-Route of infection: Through trivial wound or abrasion.

III-Predisposing factors: debilitating disease & bad general condition.

Pathology:

Site: -It is common in the face, scalp, scrotum & limbs .

- The organism invades the superficial lymphatics

Complications:

1- Toxaemia, pyaemia & septicaemia.

2- Cavernous sinus thrombosis in facial erysipelas.

3- Recurrence is very common with progressive lymphatic obstruction leading to elephantiasis.

4- Cellulo-erysipelas: over-imposed cellulitis.

Incidence:

It is common on extremes of age especially with bad general conditions.

Clinical picture:

I- Incubation period: few hours.

II- General manifestations : marked fever, headache, anorexia & malaise.

III-Local manifestations:

- Irregular rose pink patch with well defined margin.
- Small vesicles behind the spreading margin contain highly infectious serum.
- The colour fades away gradually over weeks to faint brown colour due to Hb pigment from haemolysed RBCs.

Erysipelas

Investigations: not needed (clinical diagnosis).

1- Bacteriological examination of the exudate obtained from the vesicles.

Differential diagnosis:

From cellulitis.

1- The colour is faint red and the margin is ill-defined.

2- Suppuration occurs.

3- If the face is involved the auricle is spared *(Milian's sign)* as the auricle has no SC tissue.

Treatment:

I- Prophylactic ttt :

Rapid application of antiseptics to wounds and sterile dressing.

II- Treatment of established cases:

A- General:

1- Isolation: as the disease is highly infective.

2- Antibiotic: penicillin is effective.

B- Local:

1- Rest: to decrease toxaemia & spread .

2- Lead subacetate or icthyol ointment have a soothing effect.

3- Drainage only in cellulo-erysipelas (pus).

4- Actinomycosis

Definition :

It is an infective granuloma caused by anarobic gram +ve filamintous organism.

Aetiology:

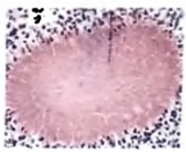
I-Causative organism : Streptothrix actinomyces israeli .

II-Route of infection: The organism lives normally in the oral cavity and alimentary tract of healthy individual. It infects the tissue through:

1- Abrasion in the mucous membrane in the presence of carious tooth.

2- Ingestion of infected material 3- Inhalation in lung.

4- Direct contamination of the wounds.



Microscopic picture

Pathology:

Site: 1- Faciocervical region (60%). 2- Abdominal (20%).

3- Pulmonary (15%). 4- Cutaneous (5%).

- The organism spreads by direct invasion of tissues and may be through bl. stream *but not lymphatics* as the organism is larger than the lumen of lymphatics.

Clinical picture :

Irrespective of the site, actinomycosis forms a firm mass that breaks down to discharge *sulfur granules* containing pus through multiple sinuses.

Cervicofacial actinomycosis

Investigations:

1- Examination of discharge:

Macroscpic examination reveals the characteristic pin head sized sulfur granules.

Microscopically→ radiating gram +ve mycelial filaments surrounded with Gram –ve clubs.

Treatment:

1- Prolonged course of antibiotics e.g. penicillin G, streptomycine & tetracycline.

2- Surgical resection of the mass or drainage of collection may be indicated to bring cure if medical treatment alone failed.

5-Madura foot

(Nocardiosis Mycetoma)

Aetiology:

I-Causative organisms: streptothrix nocardia madurae .

II-Route of infection: through cracks and fissure of the skin e.g. on walking bare-footed on infected soil or grasses as in Madurra.

Madura foot

Pathology:

Site:- the commonest is the feet, rarely the hands or other sites.

The organism spreads by direct invasion to the surrounding structures, SC. tissues, ligament, bone, joints not by lymphatics.

Incidence:

It is common in tropical regions e.g. India (Madura), Africa.

Clinical picture:

- Nodules that burst to form multiple sinuses which discharge black, yellow or red granules.
- Later on, complete disorganization of the foot may occur and the regional lymph nodes may be enlarged due to septic lymphadenitis.

Investigations:

Detection of the organism colonies in the discharge or tissue biopsy.

Plain Xray

Treatment :

1- Antibiotics: broad spectrum to control secondary infection.

2-. Penicillin, iodine & X-ray therapy were tried.

3- Dapson 100 mg *bid* for long term gives good results.

4- Surgical amputation may be indicated in disorganized feet.

6-Hydatid disease

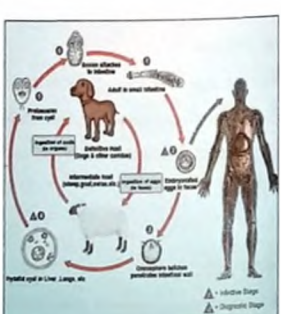
(Echinococcosis)

Aetiology:

I-Causative parasites: tapeworm Echinococcus granulosus .

II-Route of infection(life cycle):

The adult worm lives in the intestine of dogs (definitive host). Eggs pass with stool to contaminate grasses & vegetables. Sheep & cattle (intermediate host) are infected by feeding on contaminated grasses. Eggs penetrate their intestines to liver & other organs. Dogs are infected when feed on infected organs of slaughtered animals & the cycle is repeated. Man is infected as an accidental intermediate host-when contacts with dogs or



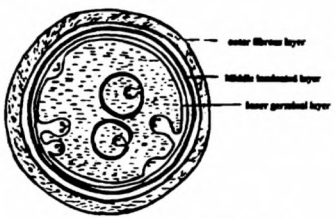
feeds on food contaminated with eggs that hatch in the jejunum to liberate the hexacanth embryo that penetrate the mucosa to the liver, lung and other organs to form hydatid cysts.

Pathology:

Site: Liver 75% Lung 15% Others 10%

Composition:

- The pathological lesion is hydatid cyst. It is composed of
  1- *Outer fibrous layer :* it belongs to the host.
  2- *Middle laminated layer* (chitinous layer).
  3- *Inner germinal layer:* with multiple budding scolices that may detach to form hydatid sand
  - Daughter and granddaughter cysts may project into or out of the mother cyst.
- -The hydatid cyst is filled with hydatid fluid.



Composition of hydatid cyst

Complications:

1-Rupture: rupture of the cyst into:

a) Liver → multivesicular cysts

b) Biliary tree → obstructive jaundice and repeated cholangitis.

c) Peritoneal cavity →peritoneal echinococcosis & anaphylaxis.

d) Pleura → secondary pleural echinococcosis.

e) Lung → bronchobiliary fistula.

2- Secondary infection: leading to abscess

3-Calcification.

Rupture of the cyst

Incidence:

-It is common in sheep breeding countries e.g. Iraq, Iran & Italy.

-It is common in people dealing with domestic dogs.

Clinical Picture:

I-Asymptomatic cases: most cases.

II-Cases present with swelling: It is characterized by:

-Rounded, painless , tense cystic swelling .

-It may show hydatid thrill on percussion due to vibration of daughter cysts against the mother cyst wall.

- Organomegaly e.g. hepatomegaly, splenomegaly ....etc.

III-Cases present with focal manifestations:

According to the site -Lung → cough, chest pain & haemoptysis.

-Bone → pain & pathological fracture.

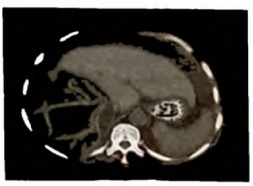
IV-Cases present with complications, e.g.. peritonitis, obstructive jaundice…etc.

Investigations :-

1-X ray : It may show calcified cyst.

2-Sonar , CT scan & MRI: to localize the cyst.

3-Casoni test: injection of 0.5 ml of sterilized hydatid fluid intradermally will produce a wheal up to 5cm in infected persons. Now, more senstive serological tests are available



hydatid cyst in the liver

Treatment:

1-Medical ttt:

Mebendazole long term therapy.

II- Surgical ttt:

Complete excision is the most effective

-Aspiration of the hydatid fluid then injection of scolicidal solution e.g. hypertonic saline or silver nitrates. The cyst is then enucleated from within the fibrous layer.

-Care should be taken not to spill the hydatid content into the peritoneal or pleural cavity and packs soaked with hypertonic saline should be tucked around the exposed part of the organ.

- The cavity may be filled with saline and closed tightly over it or packed with omentum.

-Rarely. hepatic segmentectomy or pulmonary lobectomy is needed.

*Formalin is very irritant scolicidal solution and may injure bile ducts.

Non Specific Infections

The pathological changes are not specific to a certain organism

1- Abscess الخراج

Definition:

It is localized suppurative inflammation.

Aetiology:

Causative organisms : pyogenic organisms as

Gram +VE: : Staph . Strept & pneumococci.

or Gram –VE: Gonococci, Meningococci, E. coli, Pseudomonas & Proteus.

Staph. aureus is the commonest organism as it is coagulse producer.

Pathology :

1-Stages of inflammation: due to body response to infection.

a-Hyperaemia: with intense vasodilation and increased capillary permeability

b-*Exudation*: of plasma rich in gamma globulins.

c-*Cellular infiltration*: inflammatory cells aggregate.

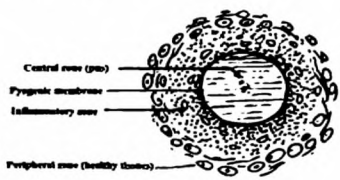
2-Stage of suppuration : Pus is formed and the abscess is formed of :

a-Central zone: contains pus.

b-Pyogenic membrane.

c-Zone of diffuse inflammation.

d-Peripheral zone of healthy tissue.



Composition of the abscess

Complications:

1-Spread: to the surrounding organs.

2-Pyaemia & septicaemia: when the organism invades the blood stream.

3-Compression manifestations.

Clinical picture:

General: fever, headache anorexia & malaise.

Local :

1-inflammation stage: dolor (pain), rubor (redness), calor (hotness) . tumor (swelling) & functio laesa (loss of function).

Subcutaneous abscess

2-Suppuration stage :

1) The fever becomes hectic.

2) Pain becomes throbbing.

3) The oedema becomes pitting.

4) Fluctuation is positive if superficial.

5) Aspiration is positive if possible.

6) Pus is discharged if ruptures.

Investigations:

1-Aspiration: through wide bore needle.

2-Imaging studies (for deep abscess): Sonar. CT, & MRI.

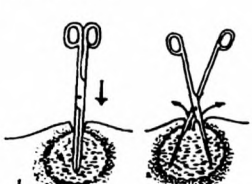
Differential Diagnosis :

From other hot , painful swelling e.g. Inflammed aneurysm.

Treatment:

I-Conservative ttt : in pre suppurative stage:

1-Antibiotic. 2-analgestc antipyretic. 3-Hot foments.



Hilton's method

II-Surgical drainage if pus is formed:

1-General or spinal anaesthesia but not local anaesthesia.

2-Incision: wide dependent drainage .In certain regions where important structures shouldn't be injured, *"Hilton's method* "is advised. The incision cuts the skin only then an artery forceps is pushed gently to enter the abscess cavity with its blades closed then open all loculi.

3-The cavity is packed for 48 hours for haemostasis.

4-Subsequent dressing are getting lighter to allow the granulation tissue to fill the cavity.

All abscesses should be incised except:

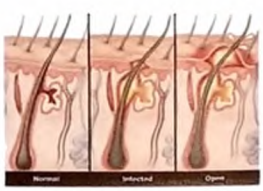
-Amoebic abscess Brain abscess Cold abscess

They should be aspirated first.

2-Furuncle (Boil) البثرة

Definition :

It is abscess of the hair follicle with perifolliculitis.



Furuncle

Aetiology:

I-Causative organisms: Staph aureus.

II-Route of infection: direct.

III-Predisposing factors: Anxiety, fatigue, DM., & debility.

Complications:

1-Furnculosis: infection of the surrounding hair follicles especially in hairy region like scrotum, cheek, axilla. It is common in patients with poor resistance.

2-Cavernous sinus thrombosis if furuncle lies in the dangerous area of the face.

Furnculosis

Treatment :

I-Conservative ttt is usually successful .

1-Antibiotics 2-Antiseptics 3-Hot applications

II-Surgical ttt, rare:

Extraction of the core.

N.B Blind boil is a healed boil without drainage but with persistent induration.

3-Carbuncle الجمرة

Definition:

It is localized infective gangrene of the skin and subcutaneous tissue caused by furnculosis spreading to the SC. Tissues.

Carbuncle

Aetiology:

I-Causative organisms: almost always staphylococcus aureus.

II-Route of infection: direct.

III-Predisposing factors: low resistance e.g.

1-D.M. 2-Ch. Nephritis. 3-general debility.

Pathology:

Site: It is common in tough skin where many fibrous septa and strands divide the dermis into compartments e.g. back of the neck, face and back.

Pathogenesis:

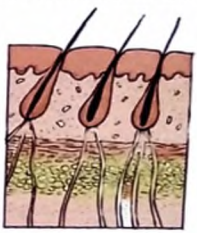
The story begins with furnculosis that extends to the SC tissue along the fibrous strands normally connecting the hair follicles to the deep fascia. The intervening tissues undergo gangrene due to

1-Pressure due to high tension inside the follicles.

2-Thrombosis of blood vessels (septic thrombosis).

3-Direct action of toxins on the tissues.

So ,the end result is an irregular multilocular lesion that is drained to the surface through multiple tracks which constitute an inadequate drainage.



Carbuncle

Complications:

I- General:

1-Toxaemia 2- Pyaemia. – Septicaemia.

II- local:

1- Spread: Cellulitis, lymphangitis & lymphadenitis.

2- Ulceration: a big ulcer due to sloughing of the overlying skin.

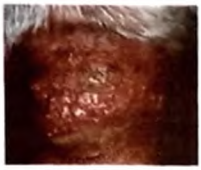
3- Cavernous sinus thrombosis if it lies in the dangerous area (triangle) of the face.

Dangerous Δ

Incidence:

Very common in diabetics & patients with chronic nephritis.

Clinical picture:



Carbuncle in the nape

General:

1- Features of the debilitating disease.

2- Fever, headache, anorexia & malaise.

3- Toxic glucosuria due to lowered renal threshold for glucose.

Local:

Early: indurated painful dusky red swelling with ill-defined margin.

Later: multiple openings discharging pus and necrotic material.

N.B fluctuation is late and difficult and not needed.

Investigations:

:Mainly to detect the predisposing factors e.g. blood sugar & renal function tests.

Treatment:

I- Conservative treatment:

1- Control of the predisposing factor.

2-Antibiotic (penicillinase resistant).

3- Hygroscopic dressing e.g. glycerin magnezia to lower tension and pain & help drainage.

II- Surgical ttt:

If medical treatment fails:

1- *Wide drainage*: wide cruciate incision with excision of all necrotic tissue and opening of all loculi to ensure adequate drainage.

2- *Excision*: Excision of the whole carbuncle if small and leave the wound opened.

4- Cellulitis (التهاب خلوى)

Definition:

It is spreading pyogenic inflammation of cellular tissue (subcutaneous tissue or fascial planes).

Actiology:

Causative organisms: streptoccci pyogenes. Rarely staph. & other organisms.

Route of infection : Direct through accidental wound or surgical maneuvers.

Predisposing factors: Low resistance e.g. DM.

Pathology:

Site: Common in the scalp, orbit, neck, limbs & pelvis. The infection spreads rapidly in the cellular tissue due to relatively poor vascularity and due to the spreading enzymes of the organism e.g. fibrinolysin, hyaluronidase ...etc.

Complications:

Cellulitis of the thigh

General:

1- Toxaemia 2- Pyaemia 3- Septicaemia

Local :

1- Spread: lymphangitis and lymphadenitis.

2- Ulceration: due to sloughing of the overlying skin.

3- Complications according to the site:

a) Cavernous sinus thrombosis: It may complicate cellulitis of the face & scalp.

b) Asphyxia: may complicate cellulitis in the submandibular region *(Ludwig's angina)* due to extension of oedema to the glottis causing stridor.

c) Osteomylitis of the skull & intracranial infection may complicate cellulitis of the scalp.

Clinical picture:

General: Fever, headache, anorexia & malaise.

Local: - Signs of inflammation e.g. pain, hotness, redness & oedema.

- The inflamed area is shiny indurated with ill-defined edge. Fluctuation is rare.

Treatment:

I- Conservative management: usually successful.

1- Big doses of antibiotic (penicillin group), anti-inflammatory drugs & hot fomentations .

3- Correction of the predisposing factors e.g. DM.

4- If no response, collection is suspected & drainage is indicated.

II-Surgical:

It is rarely needed. Multiple incisions may be required.

Necrotizing fascitis

Definition

It is a rapidly progressive nerotizing inflammation of subcutaneous tissue and overlying skin Characterized by rapid spread , significant underlying tissue damage ,severe toxemia with only minimal to moderate superficial findings

Aetiology

- Causative organism : mixed aerobic and anaerobic streptococci , staphylococci and bacteroids.
- Route of spread : direct spread from pin prick , abscess , ulcer or even surgical wound .
- Predisposing factors : 1-Low immunity 2-Obesity (excess fat) 3-Bad hygene

Pathology :

The infection proceeds into the fascial planes causing septic thrombosis of blood vessels and gangrene of subcutaneous tissue and overlying skin . the muscles are characteristically healthy .

Complications :

1- Wide spread with wide area of skin loss.

2- Toxaemia and even septicaemia .

Incidence

It is more common in diabetic and immunocompromized patient .

Clinical picture:

Earlycases:

1-Fever, headache, anorexia, malaise

2- mild superficial signs of inflammation

It needs high degree of suspicion. You can suspect it when:

* The patient's pain is greater than expected from the appearance of the wound
* The induration of SC. goes past the visibly infected area.

Early cases

Late cases:

* Multiple discolored patches develop to produce a large area of gangrenous skin.
* Initial necrosis appears as a massive destruction of the skin and subcutaneous layer.

Test incision: in the operating room (OR). Skin incision yields dirty grayish brown (dish water) exudate. If you put your finger through this incision you will find that your finger easily invades the tissue which becomes weak resistance (sweep test).

Late cases

Advanced (neglected) cases:

* Necrotic eschars that look like deep thermal burns.
* As toxins are being released, the body's organ may go into septic shock while contracting a high fever, high white blood count, and becoming disoriented. This may result into respiratory failure, heart failure and renal failure.

Neglected cases

Investigations :

TLC : leucocytosis .

Extensive necrotising fascitis of the thigh

Treatment:

[1] Surgical :

After preoperative preparation of the patient , all necrotic and infected tissue should be debrided then repeated dressings till healing or grafting .

[2] Antibiotic:

Combination antibiotic therapy covering aerobic and anaerobic bacteria is administrated before and after surgery. Clindamycin and Meropenem are ideal combination.

[3] Improve nutritional and general condition of the patient , control of diabetes .

Blood infections

1)Toxaemia.

Definition: It is merely toxins circulating in blood without organisms.

Aetiology: e.g. tetanus, diphtheria .

Clinical picture: Marked constitutional manifestations.

2) Bacteraemia.

Definition: Few non multiplying bacteria without toxins circulating in blood.

Aetiology: Low virulent organism.

Clinical picture: It is usually silent.

3) Septicaemia.

Definition: Large number of multiplying bacteria with their toxins circulating in the blood .

Aetiology:

I- Predisposing factors: Low resistance of the patient .

II- Precipitating factors: 1ry focus of infection with good venous drainage e.g., pelvic cellulitis.

Clinical picture

- Marked constitutional manifestation.
- Rigor, flushing & may be jaundice. Septic shock may develop with coma & death.

4) Pyaemia.

Definition: Septic emboli circulating in blood.

Aetiology: 1ry focus of suppuration e.g. osteomylitis, otitis media ...etc.

Clinical picture: intermittent rigor with marked constitutional symptoms & development of pyaemic abscesses elsewhere e.g. liver SC. tissue.

Hospital-acquired infections (HAI)

"Nosocomial infection"

Definition:

*infections developing after 48 hours of hospitalization or stay at a healthcare facility that was not present or incubating at the time of admission.

This can include infections that occur following discharge (usually within 4 weeks)

Aetiology:

The causative Organisms:

Resistant healthcare associated pathogens e.g. methicillin-resistant Staphylococcus aureus (MRSA), Clostridium difficile, and vancomycin-resistant enterococci (VRE)

Route of spread:

1- Patient's own flora: - Failure of correct aseptic technique.
- Contaminated surgery.

2- Indirect contact: - - Contact from hands of doctors, nursing staff, patients, visitors.
- Contaminated surfaces, e.g. door handles, cups.

3- Direct inoculation: -Surgeon or environmental flora through failure of aseptic technique.
-Contaminated instruments or dressings.
- Colonization of indwelling drains, catheters, intravenous lines.

4- Airborne spread: - Skin and clothing of staff, patients, and visitors.
- Air flow in operating theatre or ward.

5- Hematogenous spread: - Intravenous and intra-arterial lines.
- Contaminated infusions.

6- Food and waterborne.

7- Faeco-oral.

Predisposing Factors:

- Undefined colonized personnel
- Improper use of antibiotics or lack of antibiotic prophylaxis
- Central lines with parenteral nutrition
- Diabetes, malnutrition extremes of age and immunodefiency.

Pathology:

Types: - Skin
- Pneumonia
- Catheter related infection
- Surgical site infection
- Urinary tract infection
- Hospital associated diarrhea (enterocolitis)

Chapter V

General Organs

General organs is a term developed by the authors to refer to organs that share in the formation of each part of the body such as skin, muscles, nerves, blood vessels and bones.

Incidence:

* Three-quarters of nosocomial infections occur in surgical patients who account for 40% of hospital inpatients

Diagnosis:

Culture and sensitivity of wound discharge, blood, urine, stools and body secretion. Also, culture of removed central lines is done if blood borne infection is suspected.

Treatment:

Treatment of established cases

- Supportive care
- Drainage of infection
- Removal or replacement of catheters and central lines
- Control of diabetes ,and malnutrition
- Appropriate antibiotics according to hospital data e.g. vancomycin

Prophylaxis:

- Screening for carriers by frequent swab of skin and nares of hospital staff.
- Consistent hand washing or the use of alcohol-based gel
- Any patient identified as having C. difficile, MRSA, or VRE colonization or infection is then placed on contact isolation precautions (requires the use of a gown and gloves).
- Precautions for operating staff concerning aprons, gloves, glasses and goggles.
- Strict cleaning of the hospital environment
- Antibiotic prophylaxis: The aim is to reduce the incidence of surgical site infection (SSI) by preventing colonizing or contaminating organisms at the operative site.

Surgical Site Infection (SSI)

Definition:

infection occurring in a surgical wound within 30 days of operation or within 1 year if prosthesis or foreign body is implanted.

Aetiology:

*** Causative organisms** :

Most causative organisms are normal flora of skin ,pharynx & colon (The human body harbours approximately 10^{14} organisms). The commonest organisms are:

1) Streptococci, B .haemolytic, faecalis and pyogens
2) Staphylococci, aureus, methicillin resistant (MRSA), lactamase producers and Staphylococcus epidermidis (alba)
3) Escherichia coli, Salmonella, Klebsiella, Enterobacter, and Proteus
4) Pseudomonas and Actinobacter.
5) Clostridium perfringens and difficile
6) Bacteroides

Route of spread:

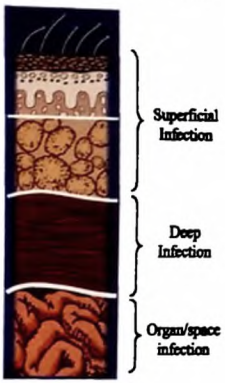
-These organisms cause infection when they move from normal residence site to abnormal site e.g. infection of colonic surgery wounds by normal flora of colon (E. coli).

- Other sources include operating theatre (inadequate air filtration, poor antisepsis) or the ward (e.g. poor hand washing compliance)

**** Predisposing factors***:

- Preoperative factors : a) bad general condition ,malnutrition and chronic diseases
  b) Septic foci elsewhere
  c) Lack of prophylactic antibiotics
  d) Poor antiseptic protocols (agents used, duration of scrubbing, etc)
- Operative factors: a) Nature of operation, time, and vascularity of tissues
  b) Hematoma and foreign bodies
  c) Perioperative bleeding, shock, hypoxia or cardiac arrest

Pathology:



****Types:***

- Superficial infection: skin and subcutaneous tissue only
- deep infection: fascial and muscle layers involved
- Organ/space infection: extension into viscera or body cavity.

Complications:

- Wound dehiscence and deep abscess formation.
- Breakdown of anastomoses and fistula formation.
- Sepsis, septic shock, and multiple organ failure.
- Need to revise vascular and orthopedic prosthesis.

Clinical picture:

- Pain, fever and toxemia
- The wound is hot, erythematous, & tender. Oedema makes stitches more tight & deep.
- Wound discharge which is mostly purulent and offensive.
- Fistula may manifest as intestinal, biliary or urinary discharge

Investigations

- CBC for leucocytosis.
- Wound and blood culture for offending organisms
- US & CT scan. Collection may be localized and aspirated

Treatment:

- Opening and debridement of the wound.
- Antibiotics
- Drainage of any collection and prevention of its recurrence by packing (*superficial infection*) and catheter drainage (*deep infection*)
- Delayed closure after granulation
- Removing prosthesis in resistant infection

Important notes

* Surgical wound with surrounding erythema and pitting oedema does not mean cellulitis. It means that there is pus within the wound that has to be drained

* Debridement (التضرير) is removal of dead, damaged, or infected tissue to improve the healing potential of the remaining healthy tissue. It may be surgical debridement or chemical debridement by use of certain solutions e.g. Eusol (Sodium hypochlorite equivalent to 0.05% - 0.1% available chlorine in an isotonic solution) or ointments e.g. Iruxol (clostridiopetidase and protease enzymes containing preparation).

Organ Transplantation

Since the beginning of the 20th century, many trials were recorded for experimental and human organ transplantation. Successful trial were only recorded in the 2nd half of that century.

Principals of organ transplantation

The transplantation team is confronted with many obstacles during their research work:

1-The source of organs, living or cadaver donors.

2-The method of preservation of organs from cadaver.

3-Immungenicity and graft rejection.

4-Problems of immunosuppressive therapy.

[1] Source of organs

[1] Living donors:

-Only for paired organs e.g. kidneys or segmental organ transplantation e.g. pancreas & liver.

-The donor should be willing and consented.

-Also, donors should be tissue-typed and cross-matched with patient (see below).

[2] Cadaver donors:

Here, the organs are removed from patients with total brain death. The criteria of total brain death are:

1-Deep coma with no response to external stimuli.

2- Bilateral dilated fixed pupils.

3- Absence of all reflexes like gag reflex, corneal reflex .....etc.

4- Inability to maintain respiration, pulse, and blood pressure for three minutes without artificial means.

5- Absent cortical activity proved by flat EEG in all channels.

[2] Preservation of the transplant organs.

There are two methods of preservation:

1- Simple hypothermia.

The organs are flushed with cool preservation solutions.

2- Continuous hypothermic pulstile perfusion.

By this methods ,organs can be safely preserved for longer periods e.g. kidney can survive for 3 days.

[3] Immunogenicity and graft rejection.

-On the surface of all somatic cells, there are cell surface antigens, which are glycoprotein molecules and act as self-markers. They are responsible for triggering the immune reaction that leads to graft rejection.

-These antigens were first recognized on lymphocytes and hence were called ***Human Leucocytes Associated (HLA)*** antigens and they were classified into HLA class I (HLA, A,B,C) and HLA class II (HLA DR, DP, DQ antigen) .

Graft rejection

Types of graft rejection

Types	Onset	Mediator	The graft	Treatment	Prognosis
Hyperacute	Immediate	Humoral	Flaccid & cyanotic	Removal	Graft loss
Accelerated	2-5 days	Humoral	Flaccid & pale	Removal	Graft loss
Acute	Within 3 months	Cellular & humoral	Oedematous and may rupture	Cortisone, ALG & OKT3	Good
Chronic	After 6 months	Cellular	Shrunken & fibrotic	Non	Bad

[4] Immunosuppressive therapy

The commonly used immunosuppressive agents are:

1- Calcineurin blockers : This group includes cyclosporin and tacrolimus .

2- Antiproliferative agents: This group includes azathioprine and mycophenolate .

3 Anti lymphocytic preparation (antibodies): This group includes :

a- Polyclonal antibodies : They are produced by sensitizing horses or sheep to human lymphocytes. Sera will contain antibodies to many clones of lymphocytes and cell antilymphocytes globulin (ALG).

b- Monoclonal antibodies: OKT3 is a monoclonal antibody to CD3 protein on human lymphocytes.

4- Steroids .

Complications of immunosuppressive therapy :

Immunosuppressive drugs increase the incidence of infection and malignancy.

I- Infection :

Opportunistic infection represents a potential risk in patients receiving immunosuppressive drugs.

A- Viral infection: The most common is cytomegalovirus (CMV) & herpes simplex (HSV).

B- Bacterial: Patients undergoing organ transplantation are at risk of wound , chest or urinary infection.

N.B. latent tuberculosis focus may be reactivated during immunosuppressive therapy.

C- Fungal infection: The commonest is candida or Aspergillus fungi .

D- Protozoal infection: The most important is pneumocystitis carnii.

II- Malignancy :

Patients under immunosuppressive therapy are at an increased risk of developing certain malignancies especially those predisposed to by oncogenic viruses. The commonest types are :

1- Skin malignancies: 50 % of transplant patients will develop skin malignancy by 20 years. The commonest is squamous cell carcinoma but basal cell carcinoma & melanoma are also common.

2- Non Hodgkin lymphoma: This type of lymphoma that develops after organ transplantation is called post-transplant lymphoproliferative disease (PTLD) .

3- Kapsoi sarcoma: Transplant patients have a 300 fold increased risk of developing kaposi sarcoma .

Renal transplantation

Indications:

End stage renal diseases that necessitates regular dialysis. These diseases include:

1- Chronic glomerulonephritis.
2- Pyelonephritis.
3- Diabetic nephropathy.
4- Hypertensive nephropathy.
5- Metabolic diseases (e.g amyloidosis, nephrocalcinosis).
6- Obstructive uropathy.

Contraindications:

1- Major co-morbid disease especially cardiovascular disease.

2- Pre-existing malignancy is an absolute contra-indication.

3- Some primary renal diseases may affect the graft such as focal segmental glomerulo-sclerosis.

N.B. Lower UTI,stricture or vesicoureteric reflux should be dealt with perliminary to transplantation .

Donors:

1- Living donors (20%): Living donor renal transplant, even between genetically unrelated individual, gives better results than well matched cadaveric grafts .

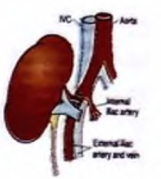
2- Cadver donors (80%): The most common source of kidney transplant. They are victims of brain death under mechanical ventilation and circulatory support (See before) .

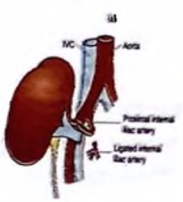
Preservation of the graft: (see before)

The ideal maximum presservation time is 48 hours

Operative technique:

- iliac incision is done with retro-peritoneal approach to the iliac vessels The kidney is then placed in the fossa and begin end to side anastomosis of the renal vein to the external iliac vein.The renal artery is anastomosed either end to side with the external iliac artery or end to end anastomosis with the internal iliac artery after ligating its distal stump.
- Implant the ureter into the recipient bladder with anti-reflux technique.





Complications of renal transplantation :

I- Techniqual complications :

1- Renal artery thrombosis (1%) and renal vein thrombosis (5%).

2- Renal artery stenosis and renal hypertension (10% of cases) present late after years.

3- Leakage from vesico-ureteric anastomosis . It is due to ischaemia.

4- Ureteric stricture: due to technical errors or ischaemic stricture..

II- Immunological complications:

1- Rejection: It is diagnosed by fever, tender kidney ,rise in creatinine and finally percutaneous biopsy.

2-Complications of immunosuppression: See before

Prognosis:

The 1 year graft survival is > 90 % and 5 year graft survival is > 80%

Liver transplantation

Indications:

Candidates for liver transplantation are cases of end stage liver cell failure due to:

I- Congenital disorders:

1- Biliary atresia. 2- Congenital liver fibrosis.

II- Trauma: Massive trauma to the liver indicating massive hepatic resection.

III- Inflammatory and autoimmune disorders:

1- Chronic active hepatitis with liver cirrhosis (the commonest) .

2- Alcoholic cirrhosis 3- Auto-immuno hepatitis . 3- Sclerosing cholangitis .

IV- Metabolic disorders:

1- Primary biliary cirrhosis. 2- Haemochromatosis .

3- Wilson's disease. 4- Familial cholestasis.

V- Vascular disorders: - Budd Chiari syndrome.

VI- Neoplasms: - Primary malignant tumours (e.g. HCC) confined to the liver.

Contra-indications:

1- Advanced cardio-pulmonary disease.

2- Active sepsis outside the hepatobiliary system.

3- Metastatic malignancy.

4- Inability of the patient to understand or withstand the implications and risk of liver transplantation.

Donors: (see before)

1- Cadaver: They are victims of brain death under mechanical ventilation and circulatory support.

2- Living donors: where only a segment is resected from the donor and transplanted in the recipient.

Preservation of the graft: (see before)

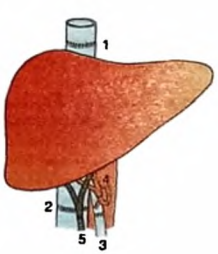
The ideal maximum presservation time is 24 hours.

Technique of the operation: There are two types of transplantation:

I- Orthotopic transplantation: The procedure is performed in three phases.

1- The dissection phase:

-In which the recipient liver is dissected from its attachments and the vessels and CBD are divided as near as possible to the liver.



2- The anhepatic phase:

- It extends from the time, the host liver is removed to the time, the donor liver is reperfused.
- The first step in the anastomosis is the cuffs of the suprahepatic (1) and infrahepatic (2) vena cava.
- Then anastomosis of the portal vein (3), after which the clamps are removed and the liver is reperfused. Then anastomose the hepatic artery (4).

3- The reperfusion phase::

- Through this phase, the blood circulates in the graft and the biliary tract is reconstructed. The gall bladder is removed and choledocho-choledochostomy , or choledocho-jejunostomy are done.

II- Heterotopic liver transplantation:

- The transplanted liver is placed beneath the recipient non resected liver .
- The heterotopic liver should have a portal inflow.

N.B. Orthotopic graft : a transplant placed in its normal anatomical site.
Heterotopic graft : a transplant placed in a site different to that where it is normally placed.

Complications:

I- Technical complications:

1- Biliary leak or stricture (5-20%).

2- Portal vein thrombosis or hepatic artery thrombosis (rare).

II- Immunological complications:

1- Primary non-function of the transplanted liver:

2- Rejection: It is manifested by abnormal liver function with characteristic histologic features.

III-Complications of immunosuppression: See before

Prognosis:

The prognosis depends largely on the original disease that had indicated the transplantation.

5 years survival rate of 80%.

Pancreatic transplantation

Indications:

The only indication is diabetes mellitus. The beneficial effect of pancreatic transplantation is :

-Obviate the need for insulin injection.

-Delays complications of diabetes.

Contraindications:

The operation is absolutely contraindicated when the recipient suffers from:

1- Coronary artery disease or myocardial infraction.

2- Chronic active infection.

3- Incurable malignancy.

Donors:

1-Cadvers donors: the whole pancreas with the duodenum are procured.

2- Living donors: only a vascularized segment is obtained.

Preservation of the graft: (see before)

The ideal maximum presservation time is 12 hours.

Technique:

There are two major procedures to drain the exocrine secretion of the pancreatic graft:

1- Enteric drainage : anastomosing the duodenal loop with small intestine.

2- Urinary drainage: anastomosing the duodenal loop to the urinary bladder to excrete the pancreatic secretion. It has the advantage of simplicity and lower infection rate.

Complications:

1) Leakage, intra-abdominal sepsis and haemorrhage from the anastomotic line.

2) recurrent cystitis, prostatitis, urethritis and urinary tract infection (due to pancreatic amylase).

Prognosis:

The 1-year graft survival is about 75%.

Small bowel transplantation

Indications:

The definitive indication is *intestinal failure* defined as impairment of intestinal function to the extent that necessitates long-term parentral nutrition. Causes of intestinal failure are:

1- Short bowel syndrome after excessive resection of the gut.

2- Intestinal motility disorders such as total intestinal aganglionosis and visceral myopathy .

3- Inflammatory bowel conditions such as necrotizing entrocolitis and Crohn's disease.

Donors:

1-Cadaver donor: the whole small gut preferably with the right colon are procured from the cadver.

2- Living donor: only a vascularized segment is obtained.

Preservation of the graft: (see before)

The ideal maximum presservation time is 10 hours.

Operative technique:

- Venous anastomosis is established by anastomosing the superior mesenteric vein of the graft to the side of the recipient portal vein.
- Arterial anastomosis is performed between the superior mesenteric artery to the native aorta.
- Then the proximal end of the intestinal graft is anastomosed end to end to the recipient jejunum or duodenum, while the distal end is usually brought out as an end stoma and the native colon is anastomosed end to side to the donor intestine.

Isolated small bowel transplantation

Complications:

I- Technical complications:

1- Intestinal leakage from the anastomosis (rare), leading to intestinal fistula .

2- Vascular thrombosis: It usually denotes graft loss.

II- Immunological complications:

1) Graft rejection.

3) Complications of immuno-suppression. (see before).

Prognosis:

The 1-year graft survival rate is about 60%. The 3-year graft survival rate is about 40% .

Heart transplantation

Indications:

End stage cardiac failure with predicted survival without transplantation is only 6-12 months due to:

1) Cardiomyopathy (idiopathic dilated cardiomyopathy) .

2) Ischaemic heart failure after massive myocardial infarction.

3) Valvular diseases.

4) Congenital anomalies e.g. Fallot's tetralogy, Eissenmenger's syndrome.

Donors:

Cardiac donors are only cadavers and should be below the age of 45 years.

Preservation of the graft:(see before)

The ideal maximum presservation time is 6 hours.

Operative technique:

- Median sternotomy is performed and the patient is put on cardiopulmonary bypass and cooled to 28ºC .
- The aorta and pulmonary artery are clamped and transected and the heart is excised through the mid atrial level leaving only atrial cuffs containing the ostea of the two vena cava on the right and the four pulmonary veins on the left .
- The donor heart is removed from ice and an incision is made on the posterior wall of both atria connecting the ostea of the pulmonary vein and the vena caval wall to prepare the flaps for anastomosis
- Left atrial anastomosis is performed first then the right using continuous sutures then aortic and pulmonary artery anastomosis are performed.
- The transplanted heart begins to function supported with atrial pacing or inotrops.

Complications:

1- Graft rejection: It may be symptomless and diagnosed be endomyocardial biopsy.

2- Accelerated coronary antherosclerosis (ACA).

Prognosis:

The one year graft survival is 85% and the five-year graft survival is 70%.

Lung transplantation

Indications:

End-stage respiratory failure that limits life expectancy less than 2 years without transplantation due to:

1) Pulmonary fibrosis.

2) Long standing pulmonary hypertension.

3) Cystic fibrosis of the lung and extensive pneumoconiosis (Asbstosis , bysinosis, silicosis).

4) Suppurative lung diseases (chronic resistant to treatment)

Donors:

Like that for heart transplantation. Single lung transplantation may be performed from a living donor.

Preservation of the graft: (see before)

The ideal maximum presservation time is 6 hours.

Opeative technique:

- After cardiopulmonary bypass, the recipient lung is removed .
- The donor pulmonary veins on a left atrial cuff are anastomosed to the recipient left atrium .
- Then the bronchial anastomosis and pulmonary artery anastomosis are carried out.

Complications:

1) Dehiscence of tracheal anastomosis.

2) Bad function of the transplanted lung. It is due to oedema and infection.

3) Tracheal stenosis:At the anastomotic line due to ischaemia and treated with dilatation.

5) Rejection : Early, it is asymptomatic diagnosed only by bronchoscopic biopsy Later, both ventilations and perfusion are affected causing serious functional deterioration and disturbance of bl. gases.

Prognosis:

The 1 year and 5 years graft survival rate for lung transplantation are 75% and 40% respectively.

Bone marrow transplantation

Indications:

The only indications in the present time are aplastic anaemia and leukaemia.

Operative technique:

1- Autotransplantation: Retransfusion of the patient's bone marrow after treating it with alkylating agents.

2- Allotransplation: It may be associated with rejection.

Endocrine cell transplantation

Pieces (slices)of thyroid, parathyroid and adrenal are transplanted into the muscles of the upper limb to get new blood supply.

Islets cells of pancreas were successfully implanted into the liver or spleen of diabetic patients but all these allografts are under trial.

The Skin & subcutaneous tissues

-Skin is the largest organ in the body.

-It weighs about 15% of total body weight

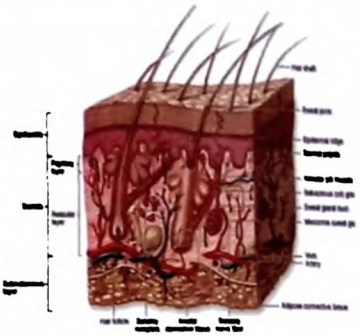
-It is a vital organ that provides a protective shield of all body organs, acts as a barrier against organisms, regulates body temperature and prevents loss of fluids from the body besides many other vital functions.

-Skin is formed of:

*Epidermis a superficial cellular layer.

*Dermis a tough deeper layer.

*Hypodermis actually it is the SC. Tissue.



Structure of the skin

I-Inflammatory conditions

A-Acute inflammations:

1- Abscess	2- Furuncle	3- Carbuncle	4- Herpes simplex & Zoster
5-Cellulitis	6- Erysipelas	7-Impetigo	8-Infected cyst

B-Chronic inflammations:

1-TB. of the skin	2- Condylomata lata ($)	3 Hydradenitis suppurativa	4- Chronic abscess
5- Scleroderma	6- Pyoderma gangrenosa	7- Condylomata acuminata	8- Radiation dermatitis

Hydradenitis suppurativa:

- It is a chronic inflammation of the apocrine sweat glands in axilla, groin & perinium.
- The caustive organisms are usually staph., strept and bacteroids
- Inflammation leads to induration, fibrosis with formation of multiple abscesses and sinuses
- Treatment includes antibiotics & muliple incisions ,but excision & grafting may be needed

Hydradenitis suppurativa

II- Cysts

1-Sebaceous cyst	2-Dermoid cyst	3-Ch. abscess	4-Cold abscess
5-Simple ganglion	6-Bursa & bursitis.	7-Haemangioma	8-Lymphangioma

1-Sebaceous cyst

Definition:

a retention cyst of a sebaceous gland due to obstruction of its punctum by inspissated secretion

Pathology:

Site : It is common in the scalp, face & scrotum .Never in the palm or the soles (thick skin contains no sebaceous glands)

Composition: It is lined with stratified squamous epithelium and contains dirty white greasy material.

Common sites

Complications:

1-Infection leading to abscess formation.

2-Ulceration: Rupture leads to malodorous ulcer resembling carcinoma and had the false name " *Cock's peculiar tumour* "

3-Sebaceous horn: due to accumulation of dried sebaceous secretions that escape slowly through the punctum.

4-Localized alopecia: due to pressure atrophy on hair follicles.

5-Malignant changes: (sebaceous carcinoma) are very rare.

6-keratoacanthoma: is considered now as an overgrowth of the cyst

Cock's peculiar tumour

Clinical picture:

-Slowly growing painless cystic swelling that has a smooth surface and a well defined edge.

-It is fixed to the skin especially at the punctum of the gland.

-It is not fixed to deeper structures unless infected.

-It is usually tense cystic (felt firm) but sometimes pressure on the cyst leads to expression of sebum from the punctum → indentation & the cyst becomes lax cystic.

Sebecous horn

Treatment:

-Excision through an elliptical incision including the punctum.

-Infected cyst is drained like an abscess then excised later if it recurs.

2-Dermoid cyst

Definition:

A cyst lined with st. sq. epith and contains dermal structures e.g. hair follicles, seb. gland … etc

Pathology:

Types:

A-Sequestration cyst

Actiology:

Inclusion of portions of surface epithelium along the line of fusion during embryonic life.

Pathology:

Site, at lines of fusion:

-Outer angle of the eye (external angular dermoid). It is the commonest site. Also, root of the nose (internal angular dermoid) & around the ear (preauricular).

-Midline of the trunk and neck (submental, sublingual &superasternal).

-Never in the limbs (no lines of fusion).

Structure:

- It is lined with st. sq. epith. and contains sebaceous material.
- In the head, it may indent the underlying bone and may be connected to the dura mater through a skull defect.

Common sites

Clinical picture:

-A hemispherical lax cystic swelling with smooth surface

-Although congenital in origin, it may not be noticed except after years when the cyst distends.

-It is painless, not tender & not attached to the skin unless infected

-It may give transmitted impulse on cough, if it is connected to the meninges.

B-Tubulodermoid

They arise from embryonic tracts (tubes):

1-Thyroglossal cyst from thyroglossal duct.

2-Branchial cyst from cervical sinus.

C-Implantation dermoid

Aetiology:

Traumatic displacement of part of the epidermis into the SC. tissue, usually puncture wounds.

Pathology:

Site, anywhere, but commonly in the fingers, palms, soles & cornea.

Clinical picture:

- It is common in manual workers.
- It forms a small tense cystic swelling. Later, it may cause pain due to pressure on nerves especially on the finger pulps.

D-Teratodermoid

Aetiology: It is a benign tumour (benign teratoma).

Pathology:

Site: common in the ovaries, testis, sacrococcygeal region & post. Mediastinum.

Structure: The cyst is lined with st. sq. epith and contains hairs, teeth, bone, cartilage or glands (derivatives of the three embryonic layers).

Treatment: the only line of treatment of all types of dermoid cysts is excision.

III-Tumour like conditions

1-Wart: (المنطة)

Aetiology: localized epithelial proliferation due to viral infection.

Clinical picture: - Small grayish horny projection that is usually multiple.

- It is common in the dorsum of the hands & feet.
- Only the plantar warts are painful.

Warts

Treatment: - Surgical excision with diathermy.

- Cryosurgery by freezing the wart using liquid nitrogen.

N.B. Condylomata acuminata are giant warts transmitted by sexual contact .

2-Callosity: (كالو= جلد سميك)

Aetiology:

localized skin thickening due to repeated friction & pressure.

Clinical picture:

- Area of thickened skin which are painless, yellowish plaques, elevated above the surface

Callosities

Treatment: - Removal of the cause.

-Shaving with sterile razor or application of keratolytic agent e.g. salicylic acid .

3-Corn: (مسمار القدم = عين سمكة)

Aetiology: neglected callosity with downgrowth of hard horny plug into the corium pressing on sensory nerve endings.

Clinical picture:

- Circumscribed conical mass with depression in the center which is very tender.

Treatment: excision.

Corn

Chapter IV

Organ transplantation

4-Keloid: (جدرة)

It is excessive amount of vascular connective tissue that extends beyond the original wound to invade the surrounding tissues. Later, it matures into fibrous tissue.

Aetiology : unknown but, it is common in:

a) Negroes b) Pregnant c) TB. patients.

-It may follow any type of wounds especially burn & vaccination wounds.

-It is commoner over the sternum, deltoid area and ear lobule,but never in palms & soles.

-It appears as an itchy firm raised patch of CT. with *claw like processes* that invade the surrounding tissues with marked disfigurement. Later, it becomes pale and shrinks.

Differential Diagnosis:

From hypertrophied scar which :

- doesn't extend beyond the original wound.
- doesn't continue to grow after 6 months.
- doesn't recur after excision.

Hypertrophied scar

Keloid

Treatment:

-Intralesional injection of steroid (triamcinolone).

-Intralesional excision as the keloid tissue itself has no tendency for recurrence ,but the surrounding healthy tissues do. Postoperative radiotherapy may be used.

NB. No attempt for wide surgical excision because it always recurs worse than it was.

5-Keratoacanthoma (Molluscum sebaceum)

-It was thought that it is a true tumour, but its progressive then regressive course raises the suspicion of its inflammatory nature.

Keratoacanthoma

Clinical picture:

-It is common on the face of middle aged people.

-It forms a red firm rounded papule that enlarges rapidly to about 2 cm in one week then regresses slowly to heal within 3 to 6 months.There is *a keratin plug* in the center.

Treatment: excisional biopsy.

6-Pyogenic granuloma (Granuloma pyogenicum)

It was thought that it is a true tumour but actually it is a mass of infected granulation tissue.

Clinical picture:

-It is common in the face, fingers & toes.

-It forms a small red mass of granulation tissue not covered by epithelium and the surrounding skin may be macerated.

Pyogenic granuloma

Treatment: excision.

7- Haemangiomas and vascular malformation

- Hemangiomas have proliferative then involutional phases while vascular malformations or hamartomas grow proportional to body and skin growth without involution.

A) Haemangioma

1) Strawberry naevus: (haemangioma simplex, infantile haemangioma).

Pathology:

- Site: commonly on the face, rare on the trunk or limbs.
- Structure: bright red mass of capillaries lined by embryonic endothelium.

Clinical picture:

-It appears shortly after birth (1-3 weeks) and increases in size in the first year of life, then usually regresses and may disappear within the first 8 years.

-It is slightly elevated above the skin.

-It may be complicated by infection ulceration ,and bleeding

Strawberry type

Treatment

- Oral predinsolone at a dose of 2 -3 mg/ kg/day
- Oral propranolol at a dose of 2 -3 mg/ kg/day
- Corticosteroid local injection.
- Resistant cases may require plastic correction.

Salmon patch

2) Salmon patch:

It is present at birth over the forehead or in the occiput and disappears at 1 year age

B) Vascular malformations.

1) Portwine stain:

Pathology:

- Site: commonly in the face & neck. It follows the distribution of trigeminal nerve.
- Structure: Condensed dilated capillaries lined with adult endothelium

Clinical picture:

-It is dated since birth. It has stationary course i.e. it doesn't vary in size apart from the normal growth & development of the skin. It is not elevated above the surface.

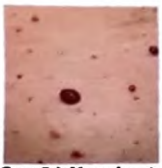
Treatment: difficult

-Excision & grafting.

-Crucyosurgery, laser ablations.

Portwine type

2) Campell de Morgan's spots :


Campell de Morgan's spots

Pathology

*Site: abdomen & chest

Clinical picture

- Small bright red spots in middle aged persons.
- It was thought that these spots precede the development of internal malignancy (not proved).

Treatment: No ttt

Osler's disease

3) Osler's disease (Hereditary haemorrhagic telangectasia):

Pathology

Site: skin & mucous membrane of the GIT.

Clinical picture:

It usually presents with unexplained anaemia with occult bl. in stool.

Treatment

Endoscopic coagulation of bleeding gastric haemangioma.

Spider naevi

4) Spider naevi:

Pathology

*Site: in the skin drained by the superior vena cava.!!

*Structure: Dilated arterioles radiating from a central one

Clinical picture

-They appear in patients with liver cell failure and may be due to oestrogen or other vasoactive substance. Compression of the central arteriole causes blanching of the radiating arterioles.

Treatment: No treatment.. only control of liver cell failure.

5) Venous malformation (*old name, cavernous haemangioma*)

Pathology

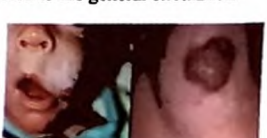
*Site: - cheek, lip (macrocheilia) & tongue (macroglossia).

-Internal organs e.g. liver, spleen, lung, colon.

*Composition: It is composed of dilated venous spaces connected to the general circulation.

Clinical picture

- Bluish, soft, compressible swelling with lobulated surface & ill-defined edge.
- It may cause troublesome bleeding (external or internal)
- Progressive development without involution.


Venous malformation

Treatment

1- Sclerotherapy: intravascular injection of sclerosing material. Repeated injection is needed

2- Gel foam embolization of the feeding vessels through angiography

3- Surgical excision: in circumscribed lesions or ligation of the feeding vessels in large lesions.

6) Arterial malformation (Cirsoid aneurysm) - old name arterial haemangioma

Pathology

* Site: commonest in the temporal or occipital region of the scalp.
* Structure: It consists of dilated tortuous arterioles (cirsoid = varix like) communicating with the general circulation and sometimes it has intracranial extension.

Clinical picture

Large, pulsating, compressible swelling in the scalp with palpable thrill and audible bruit.

Cirsoid aneurysm

Investigations

1-Plain X-ray: underlying skull defect.

2-Angiography: detects the feeding vessels & intracranial extension.

Treatment

-Ligation & excision if possible.

-Simple ligation or embolization of the feeding vessel.

8- Lymphangioma

They are hamartomas with excessive haphazardly arranged lymphatic vessels

Types:

i) Capillary lymphangioma (Lymphangioma simplex)

Pathology

*Site, in the skin or mucous membrane, especially the tongue.

* Structure, it is formed from dilated lymphatic capillaries.

Clinical picture

-Small brownish papules in skin or mucous membrane.

- Exam. with a lens reveals small vesicles.

Capillary lymphangioma

Treatment

Surgical excision.

ii) Cavernous lymphangioma

Pathology:

*Site: common in the lip (macrocheilia) & tongue (macroglossia).

*Structure: It consists of multiple intercommunicating cystic spaces that contain lymph and are lined with endothelium.

Macroglossia

Clinical picture:

Soft, translucent, cystic mass (lymphatic cyst) that may cause gross enlargement of the part

Treatment:

Surgical excision

iii) Cystic hygroma

Aetiology:

Sequestrated part of a foetal lymphatic sac e.g. jugular lymphatic sac.

Pathology:

*Site: common in the neck, axilla, mediastinum & rarely in the groin.

*Structure: like cavernous lymphangioma, but the superficial cystic spaces are large while the deeper ones are small and infiltrate the muscles.

Cystic hygroma

Incidence:

-It appears in early infancy occasionally at birth.

Clinical picture

-Large ill defined lax cystic mass in the lower part of the neck , superficial to sternomastoid muscle and may extend to the posterior triangle of the neck

-It is brilliantly translucent. It is partially compressible due to shift of fluid to deeper cysts

-It gives impulse on cough or crying .

Trans-illumination

Treatment

- Sclerotherapy using tetracycline, alcohol or others.

-Surgical excision if sclerotherapy failed. Complete ecision is usually impossible.

V- Neoplasms of skin & S.C.

A-Benign tumours

I-Tumours from epithelium: Papilloma.

Benign calcifying epithelioma.

II-Tumours from pigment cells: Benign melanoma (pigmented).

III-Tumours from the dermis: dermatofibroma.

IV-Tumours from sebaceous gland : sebaceous adenoma.

V-Tumors from cutaneous nerves: neurofibroma.

VI- Tumours from cutaneous bl. vessels.: glomangioma.

VII-Tumours from cutaneous lymphatics: cystic hygroma

VIII- Tumours from CT: lipoma.

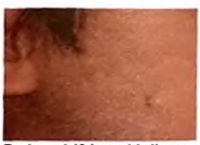
Papilloma

1-Papilloma

- They are single or multiple, sessile or pedunculated, nodular or villous and usually pigmented.
- It may have hereditary or viral aetiology (papilloma virus). Malignant changes are rare.
- Treatment: surgical excision

2-Benign calcifying epithelioma

- It is single hard mass resembling calcified sebaceous cyst.
- Most commonly in children especially in face and upper limbs.
- It is formed of mummified basal cells with calcification.
- *Treatment:* excisional biopsy.



Benign calcifying epithelioma

3-Benign melanoma

Pigmented naevus = Mole

Naevus = birth mark. Pigmented naevus = mole (شامة)

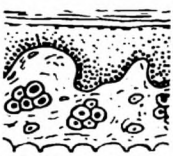
Types:

1-Simple (intradermal) naevus(the commonest):

-The melanocytes are present in the dermis.

-It may occur in any site except palm, soles & genitalia.

-It may contain hair and doesn't tend to turn malignant.



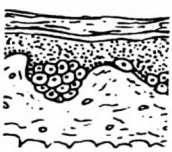
Intradermal naevus

2-Junctional naevus:

-The melanocytes are present in the basal layer of the epidermis and extend to the dermis.

-Common on genitalia, palms, soles, mucous membranes& nail folds.

-It is hairless and may turn malignant.



Junctional naevus

3-Compound naevus:

-The melanocytes are present in the dermis with junctional activities.

-It is common in infancy.

-It may be hairy and may extend over a large area of the body (giant hairy nacvus) that may give rise to metastases.

Compound naevus

4-Freckles (lentigo) (نمش):

- Melanocytes replace the basal layer of epidermis over a certain area
- It commonly occurs in exposed parts of the body especially in blonds & red haired persons.
-*Hutchinson's freckles* (lentigo maligna) is a special type that occurs in old people especially in the face and commonly turns malignant.

Freckles

6-Blue naevus:

-The melanocytes are spindle shaped and lie deeply in the dermis.

- It commonly affects face, hand & buttock.
- It is bluish in colour and may turn malignant.

Blue naevus

Treatment: Surgical excision is indicated in:

1- ugly moles for cosmesis.

2- lesions susceptible for repeated irritation e.g. in the face of men due to repeated irritaion o shaving.

3- lesions shows signs suggestive of malignant changes (see later).

4-Dermatofibroma

(Fibroma of the skin)

-It is also called sub-epidermal nodular fibrosis.

-It appears as small firm nodule.

-It may reach large size and attain malignant behavior (*dermato-fibrosarcoma protuberance*) which is a low grade fibrosarcoma.

-Wide excision with safety margin is recommended.

Dermatofibroma

5-Sebaceous adenoma

-It is a benign tumour that arises from sebaceous glands commonly in the face or scalp.

-It forms a slowly growing pinkish lobulated mass with narrow base.

-It may attain malignant behavior such as rapid growth, ulceration & recurrence after local excision (sebaceous carcinoma).

Sebaceous adenoma

6-. **Neurofibroma** (see peripheral nerves).

7- **Glomangioma (Glomus tumour)**

It is a true benign tumour arising from glomus body, which is a highly organized globular structure present in the extremities & ear. It is composed of arteriole, venule & sympathetic nerve fibers. Its function is regulation of temperature & blood flow in distal extremities

Pathology

*Site: commonest in the nail bed but may occur in glomus body elsewhere.

*Structure: It is composed of vascular, neural & smooth muscle fibres (haemangio-myo–neuroma).

Clinical picture:

- A very small tender purple spot under the nail that usually gives rise to severe attacks of pain.

Glomangioma

Treatment: Surgical excision or electrocoagulation.

8- **Cystic hygroma.** (see before).

9-Lipoma

Definition:

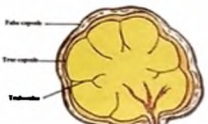
It is a benign tumour arising from adipose tissue. It is the commonest benign tumour of the body.

Pathology:

*Site: it may occur anywhere in the body where adipose tissue exists e.g. SC tissue, subfascial submucosa.. etc (see clinical types).

*Types:

1-Pure lipoma:Aggregation of fat cells that forms a yellow lobulated soft mass which has a thin true capsule that sends fibrous septa (trabeculae) which divides the tumour into lobules. Also, the tumour is surrounded by a false capsule ofcompressed surrounding tissues . The tumour gets its blood supply through its pedicle that enters it at one point.



The structure of a lipoma

2- Fibrolipoma : Lipoma with excessive fibrous tissue, so, it is firm in consistancy.

3- Naevolipoma or haemangiolipoma: Lipoma with marked vascularity, so, it is partially compressible. It is common in the parotid region.

Subcutaneous pure lipoma

Complications:

1-Compression: subthecal lipoma may compress the spinal cord leading to paraplegia.

2-Calcification: common in flexion or pressure areas e.g. axilla, groin, perineum & buttock.

3-Intestinal complications: submucous lipoma may induce intussusception, intestinal obstruction or may undergo torsion and causes considerable bleeding.

4- Infection: abscess formation.

5- Migration: SC. lipoma may migrate with gravity elongating its pedicle.

6- Malignant changes: (liposarcoma) rare and mostly in the shoulder, thigh & retroperitoneum.

Incidence

They occur in adults.

Dimpling on moving aside

Clinical picture:

There are many clinical types according to its site:

1-Subcutaneous lipoma: the commonest type

*Solitary but commonly multiple.

*Site: common in the back & shoulder

*Shape: oval or irregular

*Skin over: attached to the overlying skin by fibrous strands so moving lipoma to aside causes dimpling at multiple points.

*Size: variable from pea to melon size.

*Surface: lobulated.

*Surrounding structures: it is not attached to fascia or muscles.

* Special character: it has a slippery edge.

*Consistency: it is so soft that it usually gives pseudofluctuation.

False SC lipomas

A- Aggregated fatty tissue in certain areas e.g. neck in Cushing's disease

B-Dercum's disease (lipoma dolorosa): overgrowth of fat in certain areas of the body e.g. shoulders & thighs of fatty women (due to disturbed tissue metabolism) which may be painful or tender.

Dercum's disease

2-Subfascial lipoma: deep to the deep fascia. It is firm not lobulated and difficult to diagnose.

3-Other sites: Lipoma may arise Intramuscular, Subperiosteal, Submucosal, Subsynovial, Subserous, Subthecal or Intraglandular but never intracranially as no fat cells are present there.

Treatment:

Excision by enucleation through the plane of cleavage between the false & the true capsules. The wound should be drained.

B- Malignant neoplasms

I- Tumours from epithelium: - Basal cell carcinoma

- Epithelioma (squamous. cell carcinoma)

II -Tumours from pigment cells: malignant melanoma.

III- Tumours from the dermis: dermatofibrosarcoma protuberance.

IV- Tumours from sebaceous glands: sebaceous adenocarcinoma.

V- Tumours from cutaneous nerves: neurofibrosarcoma.

VI- Tumours from cutaneous bl. vessels: e.g. Haemangiosarcomas and kaposi sarcoma.

VII – Tumours from cutaneous lymphatics: lymphangosarcoma

VIII- Tumours from S.C. tissue liposarcoma.

1- Basal cell carcinoma (BCC)

(Rodent ulcer)

Definition:

It is a locally malignant tumour arising from the basal cell layer of st. sq. epithelium .

Aetiology:

I- Predisposing factors - Exposure to sun rays.

II- Premalignant conditions. - Xeroderma pigmentosa.

Pathology:

Site: It is very common on the face above a line that joins the angle of the mouth and lobules of the ear (seborrheic area) especially in:-

a) Nasolabial fold: b) Around the eye. c) Hair line of the scalp.

It may occur anywhere in the skin including anal canal.

Common sites

Mac: = Types

1- Nodular (Button) type.

It is the earliest lesion.

A bluish white soft nodule with dilated arterioles over it .

Nodular type.

2- Ulcerative type: (Rodent ulcer)

It is the commonest type and characterized by:-

-*Shape*: rounded, oval or irregular. -*Size*: variable.

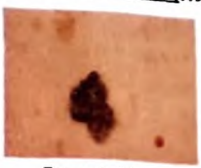
-*Edge:* is raised, rolled in (healing from margin proceeds the rate of growth) and beaded as the fibrosis (healing) divides the edge into beads.

Rodent ulcer

-*Margin:* There are telangectesic vessels.

-*Floor:* granular and may be covered with dry scab that if removed, reappears again .

-*Base:* indurated but it doesn't extend beyond margin.



Pegmented type

3- Uncommon types

a) *Invasive type:* (iceberg type): small on the surface but extensively invades the deep tissue.

b) *Pigmented type:* simulating melanoma.

c) *Cystic type:* (adenoid cystic basal cell carcinoma). It commonly occurs around eye & nose and resembles sebaceous cyst . It looks like a cyst, but not truly cystic.

Superficial spreading type

d) *Superficial spreading type* (field- fire type) flat lesion with active spreading margin.

e) *Turban tumour* (cylindroma): extensive turban (عمامة) like tumour over the scalp.

Cylindroma

MIC

Polyhedral epithelial cells arranged in masses with a peripheral raw of columnar cells with parallel nuclei giving the characteristic *palisade appearance (منظر السياج)*

Microscopic picture

Spread:

1- Local spread: It is the only route of spread. The ulcer burrows deeply in tissues (rodent ulcer) and extends peripherally.

2- Lymphatic & blood spread: it doesn't give lymphatic or blood spread. (locally malignant). Enlarged draining lymph nodes may be due to either:

a) secondary infection b) basosquamous changes.

Neglected BCC

Complications:

1- Invasion of the surrounding tissues: causing destruction of cartilage, bone & soft tissues with marked disfigurement.

2- Infection.

3- Basosquamous changes.

4- Complications of surgery & radiotherapy

Incidence:

- It is common in old & middle aged people especially males.
- It is more common in fair, freckly skin.
- It is more common in people with outdoor jobs (exposure to sun).

Clinical picture:

See the macroscopic types.

Investigations:

Excisional biopsy.

Treatment

I-Surgical ttt:

Indications = contraindications of radiotherapy.

1-Very small lesion (reconstruction is not needed).

2-Very large lesion (> 2cm) (it needs more than 2 planes of irradiation).

3-Lesion infiltrating bones (as bone trabeculae protect the malignant cells & irradiation may cause bone necrosis).

4-Lesions over cartilages or near the eye as radiotherapy is harmful to these structures (eye shields may be used).

5-Radioresistant lesions.

6-Radiorecurrent lesions.

Technique:

- Excision with about ½ inch (½ cm in the face) safety margin.

The defect after excision is closed with:

*Primary sutures if small

*Skin flaps or graft if large.

- *Mohs' micrographic surgery* = Serial horizontal excision of the tumour with frozen section exam. of each layer till it becomes cancer free. The wound is painted with mercurochrome and left to heal with secondary intention. The aim is to reduce skin loss in critical areas.

II Radiotherapy:

-Using Radium needles or external X-ray therapy.

-The dose is variable according to the size of the lesion, and is fractionated over several weeks to minimize scarring.

III Other options

1-Cryosurgery: only small lesion to minimize scarring.

2-Topical chemotherapy: 5 FU ointment (Effudex) (not accepted).

3-Curettage & electrodesiccation: for small lesions (didn't get popularity).

Prognosis

It is excellent if adequate surgical excision was done (recurrence is 0%) .

Recurrence occurs with inadequate surgical removal, bone or cartilage infiltration, or basosquamous changes.

2- Squamous cell carcinoma (SCC)
(Epithelioma = acanthoma = prickle cell tumour)

Definition:

It is malignant tumour arising from prickle cell layer of stratified squamous epithelium.

Aetiology:

I- Predisposing factors:

1- Excessive exposure to sun light .

2- Chronic irritation of skin by arsenicals, tars & hydrocarbons .

II Premalignant conditions:

1- Solar (Actinic) keratosis = Senile keratosis:

- It is due to prolonged exposure to sunlight, so it affects the exposed parts of old persons. It forms small, scaly, superficial lesion.

Solar keratosis

2- Bowen's disease:

- It may be due to solar radiation or irritation by arsenicals.
- It is considered intra-epidermal carcinoma (carcinoma in situ).
- It forms hyperkeratotic brown plaque surrounded with erythema.

Bowen's disease

3- Xeroderma pigmentosa.

-It is a hereditary (autosomal recessive) disease.

-It is characterized by excessive sensitivity to sunlight with hyperpigmentation of the exposed parts with atrophy & dryness of skin .Ectropion is common.

-High incidence of squamous & basal cell carcinoma.

Xeroderma pigmentosa

4- Irradiation dermatitis:

- Common in the face & hands of persons who work in radiation field .

5- Chronic ulcers & scar:

-Ulcers of varicose veins, old burn & sinus of ch. osteomylitis have tendency to turn malignant

-Epithelioma on top of old scar is called " *Marjolin's ulcer* " which is

Irradiation dermatitis

1) Painless as the scar has no nerves.

2) Slow growth & late metastases due to fibrosis ,but once it invades the surrounding healthy tissue it grows rapidly with pain and lymphatic metastases.

Marjolin's ulcer

6- Leukoplakia:

White thick patches over mucous membranes.

Leukoplakia

7- Lupus vulgaris:

It is tuberculosis of the skin.

8- Papillomas:see before

It rarely turns malignant.

Lupus vulgaris

Pathology:

Site: It commonly affects:

1- Exposed parts e.g. face & hands.

2- Scrotum in chimney sweepers.

3- Mucous membranes lined with stratified squamous epithelium e.g. oral cavity, pharynx oesophagus & anal canal. It may also occur on top of squamous metaplasia of other types of epithelium e.g. renal pelvis and urinary bladder.

Nodular type

Macroscopically: = Types.

1- Nodular type:

- It is the earliest lesion.
- It grows rapidly & ulcerates.

Ulcerative type

2- Ulcerative type: (classic type)

It has the following characters:

Solitary

Size : variable. *Shape:* irregular.

Edge: is raised everted due to rapid growth at the periphery that raises the edge and everts it over the surrounding tissue.

Ulcerative type

Floor: necrotic.

Base: indurated that extends beyond the visible margin of the ulcer.

3- Other types:

a) *Invasive (diffuse infiltrative) type*: It has a tendency to infiltrate deeply in the tissue.

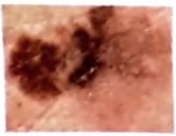
Invasive type

b) *Pigmented type:* simulating melanoma.

c) *Papillary type:* warty growth with infiltrating base. It may arise from papilloma e.g. tongue.

d) *Deep fissure:* indurated deep fissure (longitudinal ulcer).

e) *Raised plaque:* elevated patch of mucous membrane e.g. in the lip



Pigmented type

Microscopically:

1-Differentiated type: There are typical groups of malignant cells (cell nests) with keratin in the center.

2- Undifferentiated type: Sheets of malignant cells with no cell nests or central keratin.

-It is common in oral cavity, pharynx, oesophagus and bronchus.

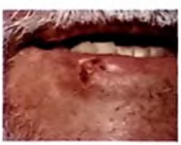
Papillary type

Spread:

1- Local spread: direct infiltration of the surrounding tissue.

2- Lymphatic spread: early.

3- Blood spread: rare & late.



Fissure type

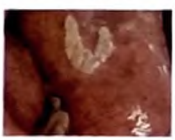
Complications:

1- Invasion of the surrounding tissues, causing destruction. obstruction & or bleeding.

2- Infection.

3- Distant metastasis.

4- Complications of surgery & radiotherapy.

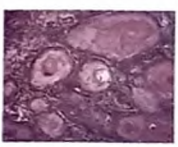


plaque type

Incidence:

-Common in old males.

-Common in persons with outdoor jobs.



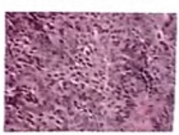
Differentiated SCC

Clinical picture:

See the macroscopic types.

Investigations:

Incisional or excisional biopsy.



Undifferentiated SCC

Treatment:

1- Surgical treatment:

Indications:

The same as in basal cell carcinoma.

Technique: Surgical excision with safety margins about 1 inch safety (only 1cm in the face).

Mohs' surgery may be used (see before).

-If the draining lymph nodes are enlarged, block dissection is indicated.

2- Radiotherapy:

- The same indications & applications for rodent ulcer.
- It gives good results, but inferior to that of basal cell carcinoma.

3- Other options:

Cryosurgery, topical chemotherapy & curettage with electrodesiccation are used in small lesions especially in elderly unfit persons.

Prognosis:

Cutaneous sq. cell carcinoma has a five-year cure rate of about 90% if adequately treated.

3- Malignant melanoma (Melanoma)

Definition:

Highly malignant tumours which arise from melanin producing cells.

Aetiology:

1- Predisposing factors:

1) Sunlight: repeated prolonged exposure to sun rays.
2) White skinned and fair-haired people.
3) Familial tendency: Familial Atypical Multiple Moles Melanoma syndrome (FAMMM syndrome).

FAMMM syndrome

2- Premalignant conditions:

1) Junctional, compound and blue navei.
2) Hutchinson's freckle.
3) Conjunctival melanosis .

Hutchinson's freckle

Conjunctival melanosis

Pathology:

Site:

Skin: anywhere in the body but common in the nail fold & bed (subungual melanoma & sole of foot (common sites in Egypt).

Mucous membrane: anal canal, conjunctiva, lips & nose.

Internal organs: arachinoid, adrenal medulla & iris of the eye.

Superficial spreading type

Macroscopically: Types:

1- Superficial spreading type: (the commonest) (65%)

A black, irregular, slightly raised lesion may be with satellites around.

2-Nodular type: (15-25%).

- It occurs more commonly in a younger age group.
- Black or brown nodule with infiltrative base, rapid growth & early ulceration.
- It has a poor prognosis.

Nodular type

3- Other types.

a) Lentigo maligna melanoma.

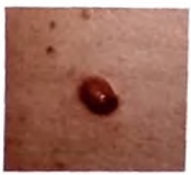
- It occurs on top of Hutchinson's freckle which is common in the face especially over the malar prominence.
- It is black or brown irregular macule with slow growth and good prognosis.

Lentigo maligna melanoma

b) Amelanotic melanoma:

-It commonly occurs in the eye, meninges & MC junction.

- It forms pinkish lesion with minimal pigmentation.
- It may give melanotic secondaries.
- It has the worst prognosis.



Amelanotic melanoma

c) Acral lentiginous type:

- It commonly occurs in the palm, soles and subungual sites.
- It has a poor prognosis.

Subungual melanoma

Microscopically:

There are two types .

1- Melanocarcinoma: the cells are rounded or polyhedral.

2- Melanosarcoma: the cells are spindle shaped.

In both types. the cells show malignant criteria and infiltrate the dermis. The melanin pigment is present intra & extracellularly.

Spread:

1- Direct spread: to the surrounding.

2- Lymphatic spread: by both permeation & embolization. Satellites & in-transit metastases are due to lymphatic permeation.

Satellites

3- Blood spread: is common to liver, lung & other organs. Heavy blood metastases may cause *melanuria* (dark urine on exposure).

In-transit metastases

Staging = classification = leveling.

There are two staging parameters:

I- Clark's staging or levels.

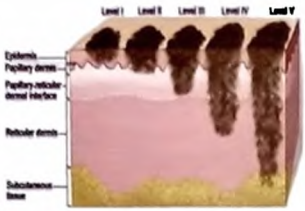
Level I: malignant cells don't invade the basement membrane.

Level II: malignant cells invade the basement membrane but didn't fill dermal papillae yet

Level III: malignant cells infiltrate up to the junction between the papillary & reticular dermis

Level IV: Malignant cells invade the reticular dermis

Level V: Malignant cells invade the subcutaneous tissue



Clark's Staging for melanoma

II- Breslow Tumour Thickness Measurement (TTM)

The thickness of the tumour is measured by an optical micrometer from the granular layer to the deepest melanoma cell. Tumours less than 0.75 mm have good prognosis

Level I: < 0.76 mm. Level II: 0.76- 1.5 mm Level III: 1.6- 2.25 mm

Level IV: 2.26 -3 mm Level V: >3 mm

Complications:

As squamous cell carcinoma.

Incidence:

- It is common in white skinned people, commoner in females than males and rare in children
- It is common in Australian and rare in Asians.

Clinical picture:

I- Melanoma arising on top of benign melanoma:

The following criteria are suggestive of malignant transformation:

1- Rapid growth. 2- Increased pigmentation. 3- Falling of hair.

4- Ulceration or bleeding. 5- Pain or pruritis 6- Induration.

7- Appearance of satellites. 8- Enlarged draining LNs .

II- Melanoma de novo

Treatment:

I- Surgery:

It is the main line of treatment as melanoma is radioresistant.

Technique:

- Wide excision with 2 inches safety margin in all directions that may include the deep fascia.
- The draining LNs are removed en block when enlarged. Radical excision (1ry tumour + draining LNs + tissues inbetween) may be done in proximal tumours of the limbs.

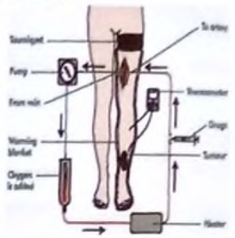
* Some surgeons prefer to delay LNs dissection for 3 weeks after removal of the primary tumour to leave them act as a filter for malignant cells in the lymphatics.

II- Chemotherapy:

-Mainly for inoperable tumours, recurrent cases & in-transit metastasis.

-Actinomycin D, Vincristine & Melphanan (phenyl alanine mustard) are used.

-Best results are obtained with *isolated limb perfusion*. A torniquet is applied to the limb and an extracorporial circulation is established with a pump and oxygenator (see the figure). By this method, melphanan may be used in high dose with little or no systemic side affects.



Isolated limb perfusion

III- immunotherapy:

Spontaneous regression may occur in some melanoma probably due to activation of immune mechanism. Stimulation of immunity is tried with interferon,levimazole, interleukin-2, and tumour infiltrating lymphocytes.Also, vaccination with BCG or small pox vaccine can be done.

Prognosis

The prognosis depends upon several factors such as:

1-The size of the lesions & depth of invasion (Clark & Breslow classification)

2-The type of melanoma: prognosis is good with lentigo maligna melanoma & superficial spreading type and poor in nodular type and amelanotic melanoma.

3-Site of the tumour: melanoma on limbs has a more favorable prognosis than that of the trunk.

7- Haemangiosarcomas

(haemangioendothelioma or haemangiopericytoma)

- It is highly malignant tumour
- It is soft bluish tumour that may occur elsewhere in the body
- It may ulcerate and bleed massively
- It is treated with combination of surgery and radiotherapy

haemangioendothelioma

8- Kaposi sarcoma

Definition:

It is a malignant tumour of the vascular endothelial cells.

Pathology:

There are two types:

1- Non aggressive form that occurs commonly in elderly persons.

2- Aggressive form that occurs in immunocompromized persons e.g. AIDS patients usually with visceral involvement, fever & lymphadenopathy.

Non aggressive form

Aggressive form

Clinical picture:

Typically, there is purplish – red flat lesions on skin or mucous membrane.

Treatment:

Chemotherapy & radiotherapy. Surgery only for surgical complications of intestinal lesions.

9- Lymphangiosarcoma

Lymphangiosarcoma

- It is highly malignant tumour that arise from cutaneous lymphatic vessels
- It is common in lymphoedematous arms after mastectomy
- It is treated with high amputation

10- Liposarcoma

Liposarcoma

- It is the commonest sarcoma in human.
- It is common in the retroperitoneum, shoulder & lower limb.
- Wide surgical excision is done whenever possible.
- The 5-year survival rate is about 50 %.

NB. The term vascular navie includes *haemangiomas. vascular malformations and true vascular tumours*

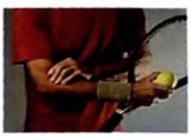
Muscles, Tendons and related structures

Sprain

Definition: Rupture or stretching of some muscle fibres due to violent muscular contraction.

1- Tennis elbow (Lateral epicondylitis):

- Sprain of the extensor muscles near their common origin from the lat. epicondyle of the humerus.
- There is pain & tenderness over the lat. epicondyle, increased with active dorsiflexion of the hand.
- It improves spontaneously after some months hastened by local injection of corticosteroids.



Tennis elbow

2- Golfer elbow (Medial epicondylitis):

- It is simlar to tennis elbow but affecting the common flexors origin from the medial epicondyle.

Golfer elbow

Rheumatic Myositis (Fibrositis)

- It is caused by exposure to draughts or dampness or secondary to septic focus.
- It is characterized by severe pain and stiffness of muscle. Pain increases with movement.
- It is common in the neck (wry neck), the back (lumbago), and shoulder.
- It is treated with antirheumatics, analgesics & short wave therapy plus ttt of septic focus.
- Local injection of corticosteroids or local anaesthetic may be of benefit.

Tenovaginitis

Definition : It is inflammation of the fibrous sheath of the tendon.

1- De Quervain's disease:

- It is stenosing tenovaginitis of the fibrous sheath of the abductor pollicis longus & ext. pollicis brevis at the lower end of the radius due to excessive use of the thumb as in typist & pianist.
- There is marked pain on abduction of the thumb and an oval swelling of the tendon is detected just above the styloid process of the radius.
- It is treated by
  - *Local injection corticosteroids
  - *Surgical division of the stenosed thickened fibrous sheath under local anaesthesia.

De Quervain's disease

2- Trigger finger:

- It is stenosing tenovagintis of the fibrous sheath of the flexors of the fingers especially the middle & ring fingers in middle aged woman.
- When the finger is flexed, the part of the tendon proximal to the fibrous sheath becomes swollen and this bulge will interfere with extension of the finger. Forceful extension of the finger will cause the finger to *snap* through the area like a pistol or trigger of the gun and hence its name.
- Treatment is similar to De Quervain's disease.

Trigger finger

Ganglion

Aetiology: -

1-Herniation of synovial membrane of joint or tendon, which becomes isolated to form a cyst

2-It may develop as myxoedematous degeneration of connective tissue in relation to tendon sheath or joint capsule (more accepted).

Pathology:

Site: common in the dorsum of the hand in relation to tendons & joints.

The wall is formed of fibrous tissue with no endothelial lining & contains clear jelly like material.

Simple ganglion

Clinical picture:

-Painless, rounded, cystic swelling in the dorsum of the hand.

-It becomes tenser and less mobile on contraction of the extensor muscles.

Treatment:

- External pressure may rupture the ganglion but recurrence is the rule.
- Complete surgical excision is the only method for permanent cure.

Student's elbow

Bursitis

Sites:

1-Olecranon bursa: *Student's elbow*: in the back of elbow.

2-Prepatellar bursa : *Housemaid's knee*, (infront of patella).

Housemaid's knee

3-Semimembranosus bursa in the popliteal fossa:

-It lies in the medial part above the joint line

-It should be differentiated from Baker's cyst which:

*occurs in older age.

*occurs on top of osteoarthritis of knee with evidence of knee effusion.

* lies in the center of the popliteal fossa below the joint line.

Semimembranosus bursa

Treatment:

surgical excision.

Dupuytren's contracture

Definition :

Idiopathic disease characterized by progressive thickening & shortening of the palmar aponeurosis.

Aetiology:

Unknown but repeated trauma and chronic inflammation were suggested as a possible aetiology, but it appears to be a collagen disease.

Pathology:

There is progressive thickening & contracture of the plamar aponeurosis leading to flexion deformity of the fingers.

Site: -It usually begins in the medial side of the aponeurosis

-It is bilateral in 50% cases.

-It rarely affects the feet as weight bearing prevents the flexion deformity of the toes.

Complications:

Disability due to flexion deformity of the fingers & stiffness of joints.

Incidence:

-It is common in

* Alcoholic *Phentoin ttt. * Cirrhotic *Diabetic

-It is commoner in young adult manual worker males (♂:♀= 10: 1).

Clinical picture:

-The condition starts as a nodule which appears at the base of the ring or little finger followed by flexion contracture of these fingers which increases progressively untill the tips of the fingers press firmly against the palm.

- The distal interphalangeal joint is free.

- Flexion of the wrist doesn't improve the flexion deformity.

Differential diagnosis:

From other causes of flexion deformity of the fingers.

I- Congenital : - Congenital short palmar aponeurosis .

II- Traumatic :- Burns of the palm.

III- Inflammatory :

1- Tenosynovitis of the flexor tendon.

2- Advanced stage of rheumatoid arthritis.

IV- Vascular:

1-Volkmann's ischaemic contracture

V- Neurological:

1- Combined ulnar & median nerve injury.

2- Klumpké paralysis.

3- Spinal cord lesions: syringomyelia & poliomyelitis .

VI-Collagen disease :

-Dupuytern contracture .

rheumatoid arthritis

Volkmann's ischaemic contracture

Treatment :

I- Conservative ttt: For early cases

It includes physiotherapy, symptomatic ttt .

II- Surgical ttt: For late cases

It includes:

1- Subcutaneous fasciotomy .

2-Excision of the palmar fascia, capsulotomy of stiff joints & skin graft to straighten the fingers

Neoplasms

A- Benign tumours

Lipoma, fibroma & haemangioma may arise from muscle.

B- Malignant tumours

1- Recurrent fibroma of Paget

(Desmoid tumour)

Definition:

It is a local malignant tumour arising from the fibrous sheaths (rectus sheath).

Aetiology:

1- It usually follows trauma: stretch of muscle fibres, small haematoma & operation scar.

2- It is common in Gardner's syndrome (see tumours of the colon).

Pathology:

Site: It arises in the rectus sheath below the level of the umbilicus.

It always arises to one side (usually the right.) never in the midline.

Mac: -It forms a pinkish hard non-capsulated mass.

- Cut section shows parallel fibres like tendons and hence the name ***"Desmoid tumour"***

MAC

Mic: - It resembles fibroma but with giant foreign body cells.

Spread: It spreads only locally; early in the mother muscle then infiltrates the surrounding.

Complications:

Infiltration of the surrounding tissues may be down to intraperitoneal structures.

MIC

Incidence:

It most commonly (80%) occurs in multiparous females.

Clinical picture:

- It represents as slowly growing painless mass in the lower abdomen with nodular surface, hard consistency and ill-defined edge.
- It can be moved slightly from side to side and becomes fixed on contraction of the muscles.

Investigations: -

1- CT scan & MRI.

Treatment:

Only by wide excision with one inch safety margin including the whole thickness of the muscle and the defect in the abdominal wall is repaired with proline or nylon mesh.

2- Fibrosarcoma

Definition:

It is a spindle cell sarcoma.

Aetiology:

Cell of origin: fibroblasts of deep fascia, aponeurosis & periosteum of bone.

Pathology:

Site. The commonest site is the thigh then trunk and upper limb.

MAC: - Rounded or nodular mass, pale gray in colour with variable consistency (soft, firm) acco-rding to the amount of collagen fibres. It may have a false capsule from the surrounding.

MIC: *1- Differentiated tumours* show parallel typical fibroblasts separated with plenty of collagen fibres.

2- Undifferentiated tumours: show larger cells which are crowded together with marked malignancy & less collagen in between.

Spread

1- Direct spread: to the surrounding structures.

2- Blood spread: to the lung.

Complications:

1- Infiltration to surrounding tissues. Blood vessels are usually compressed rather than infiltrated.

2- Distant metastasis.

Fibrosarcoma

Incidence:

- It represents about 15 % of sarcomas.
- It is more common in the young adults.

Clinical picture:

- Differentiated type is a slowly growing, firm; non-tender mass usually takes the shape of the mother muscle. Early, it is mobile from side to side but later ,it becomes fixed.
- Undifferentiated type is a rapidly growing tender mass with variable consistency. It tends to infiltrate the superficial structures first.

Investigations:

1- CT & MRI.

Treatment:

1-Wide surgical excision including the whole muscle & wide safety margin.

2- Amputation may be needed in late cases.

CT picture

3- Rhabdomyosarcoma

- It is a sarcoma of striated muscle fibres.
- It may arise in the oral cavity, neck, vagina & peritesticular region (good prognosis). When it arises from the nasal sinuses, middle ear, extremities, it has a poor prognosis.

The Veins

Anatomy of the veins of the lower limb

The lower limb is drained by the following venous system:

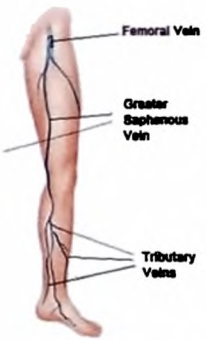
I- Superficial system: (Superficial to deep fascia).

It includes:

1- Greater (long) saphenous vein:

It begins at the medial end of the dorsal venous arch of the foot and ascends infront of the medial malleolus to the medial aspect of the leg then behind the knee to the inner aspect of the thigh till the saphenous opening (1.5 inch below & lateral to the pubic tubercle where it arches to join the femoral vein.

N.B. 1/3 of population has double saphenous veins.



Greater saphenous vein

* *Major tributaries of the greater saphenous vein:*

I- In the thigh :

1- Superficial circumflex iliac vein.

2- Superficial epigastric vein.

3- Superficial pudendal vein .

4- Posteromedial vein .

5- Anterolateral vein .

II- In the leg :

1- Posterior arch vein.

2- Anterior vein of the leg.

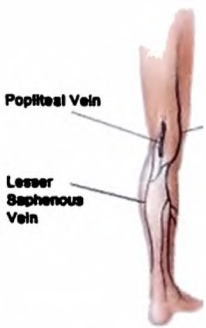
3- Tributaries from the short saphenous vein.

III- In the foot :

The dorsal venous arch.

2- Lesser (short) saphenous vein :

It begins at the lateral end of the dorsal venous arch and ascends below then behind the lateral malleolus to run along the lateral edge of tendon Achilles in the posterior midline of the leg to the middle of the popliteal fossa where it pierces the deep fascia to join the popliteal vein.



Lesser saphenous vein

II- Deep (system) veins: (deep to the deep fascia) .

It includes:

1- Vena comitants of the anterior tibial, posterior tibial & peroneal arteries .

2- Popliteal vein .

3- Femoral vein .

4- The intramuscular veins that tends to form venous sinuses e.g in the soleus muscle that drains into post. tibial & peroneal vv.

Deep system veins

III- Communicating veins (Communicators):

They are the veins that join the superficial veins with the deep ones. They comprise:

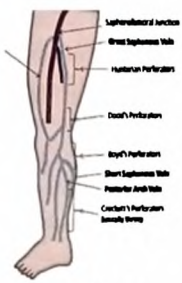
1-Sapheno-femoral junction.

2-Sapheno-popliteal junction.

3-At the midthigh (Hunterian communicators).

4-Just above the knee (Dodd's communicator)

5-Just below the knee (Boyd's communicator).



Communicators

IV -Perforating veins: (Cockett's perforators)

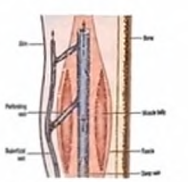
They connect small subcutaneous venules to the deep system directly. They have little relation to the saphenous system. They are present in the region of the ankle. They include:

1-Three medial ankle perforators: lie 2,4,6 inches above the medial malleolus. They connect small venous arches at the lower end of the posterior arch vein to the posterior tibial vein.

2-One lateral ankle perforator: lies at the junction between the lower 1/3 with the upper 2/3 of the leg. It communicates a tributary of the short sapenous vein to the peroneal vein.

Medial perforators

*All the veins are provided with bicuspid valves except the venous sinuses in the calf muscles. These valves are unidirectional and permit the flow of blood only in one direction towards the heart i.e from superficial to deep & from below upward . This helps the flow of blood from the lower limbs to the heart.



Valve system

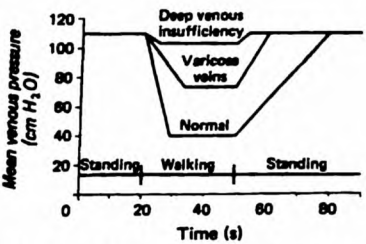
The highest valve of the lower limb lies in the femoral vein below the inguinal ligament.

Physiology

-The muscles of the lower limb together with the competent-valved veins act as a functional pump that pumps blood back to the heart. When they contract inside the tough fascial compartment they squeeze the deep veins and push the blood upward and when they relax, the pressure drops in the veins, so, they suck the blood from the superficial system and so on. The valves ensure the unidirectional flow and prevent the regurge of blood from above downwards or from deep to superficial veins. This muscular pump is called *the peripheral heart.*

-For this fact, the pressure drops in the superficial veins of the lower limb during walking or exercise and returns gradually to the pre-exercise level when walking stops.

-The pressure in a vein on the dorsum of the foot in erect position equals the distance from the right atrium (the effective zero level) to the foot i.e. 100 - 120 cm H_2O which normally drops to about 30 cm H_2O during exercise and returns gradually to the pre-exercise level when exercise stops.



Measuring the venous pressure in dorsal venous arch of the foot before, during and after excercise

-In primary varicose veins the valves of the comunicators are incompetent allowing some regurge of blood into the superficial veins but don't markedly disturb the muscular pump.

-In secondary varicose veins, the valves of the deep vein are incompetent and so impairing the pumping action of the muscle and as a result the pressure in superficial veins fails to drop or even increases during exercise (walking venous hypertension).

Venous thrombosis

1-Superficial vein thrombosis

Aetiology

It is caused by damage of intima by trauma or inflammation:

1. Trauma: repeated injection, irradiation, and irritant solutions.
2. Inflammation: -Thrombophlebitis of VV.
   -Thrombophlebitis migrans in Burger's disease or deep seated malignancy.

Clinical picture

The thrombosed vein feels like tender cord with surrounding redness & mild oedema.

Treatment:

Conservative ttt: - Bandage - Immobilization - local anti-inflammatory cream.

Surgery : It is indicated only in greater saphenous thrombosis → Trendlenburg's operation.

Deep venous thrombosis (DVT)

Definition:

Thrombosis of the deep veins (deep to the deep fascia).

Aetiology:

Causes = Virchow's triad

1-. Change in the wall of the vein in the form of intimal damage.

2-. Change in the blood flow in the form of stasis.

3-. Change in the blood composition in the form of hypercoagulability.

Predisposing Factors:

1- Operations, 50% of cases are postoperative especially after pelvic and septic operations.

2- Pregnancy, malignancy, & recumbancy.

3- Senility, obesity, viscosity (e.g. polycythaemia)

4- Hypercoagulable diseases e.g. protein C & S defeciency

Pathology:

Site: commonest in the calf veins – iliofemoral – I.V.C.

Types: there are two different types:

	Phlebothrombosis	Thrombophlebitis
- Aetiology	-Thrombosis due to stasis	-Thrombosis on top of inflammation
-Incidence	-common	-rare
-Site	-usually in the calf veins	-usually in the iliofemoral veins
	-It occurs on healthy vein	- It occurs on diseased vein
-1ry thrombus	-small enlarges & propagates	-large with little propagation
-Embolism	-loose, detaches easily → pul. embolism	-adherent unless infected → pyaemia

Complications:

I- Early complications:

1-Pulmonary embolism: due to detached thrombus impacted in pulmonary art. or its branches.

2-Venous gangrene: occurs due to complete obstruction of venous return from the lower limb.

II- Late complications:

1-Secondary varicose veins due to:

a. Incomplete canalization of deep system. b. Damaged valves.

2-Chronic venous insufficiency: (postphlebitic leg): It follows iliofemoral thrombosis

characterized by walking venous hypertension, leg ulceration, & lipodermatosclerosis.

Incidence:

Common in risky groups mentioned before e.g. postoperative, pregnancy – etc

Clinical picture:

I- Silent cases:

Most cases are silent (up to 85%).

II-Typical cases:

1. Calf veins thrombosis (most common) :

-History of operation (3rd or 4th postoperative day) or other predisposing factors.

-General: fever & disproportionate tachycardia.

-Local: *Tense tender calf.

* Mild ankle & foot oedema.

**Homan's sign*: dorsiflexion of the foot causes pain in the calf due to stretch of the calf veins. It is painful, not reliable, nonspecific and risky i.e. may dislodge thrombus to circulation.

2. Iliofemoral thrombosis:

-It may be due to extension of calf vein thrombosis (common) or due to thrombophlebitis secondary to pelvic inflammatory lesion.

-There are two clinical types or forms:

a) Phlegmasia alba dolens which it is the less severe form:

* it is associated with arterial spasm (due to lymphangitis)

* There are pallor, coldness & weak pulsation.

b) Phlegmasia cerulae dolens which is more severe form:

* There is almost complete obstruction to venous return.

* There is cyanosis, marked oedema then venous gangrene.

Phlegmasia cerulae dolens

IVC thrombosis

3. Inferior vena cava thrombosis:

There is marked oedema of both lower limbs and scrotum or vulva with dilatation of collaterals between IVC & SVC such as:

`From below { Superficial epigastric vv. / Superficial circumflex iliac vv } with { Superior epigastric .vv / lateral thoracic vv. } from above

The direction of flow is upward determined by two fingers emptying & filling test

III- Cases may present with complications

Cases may present with pulmonary embolism , postphlebitic leg or secondary VV.

Investigations:

1- Ascending venography (obsolete):

2- Doppler U/S: (accuracy is about 85%):

- Normally, when U/S probe is put on the femoral vein, the venous flow gives continuous venous hum and on squeezing the calf, a roaring sound is heard due to exaggeration of flow.
- In iliofemoral thrombosis, the venous hum is absent and in popliteal vein thrombosis the roaring sound is absent.

3- Duplex U/S: (accuracy reaches 95%):

- It combines vessel imaging and flow study resulting in high diagnostic accuracy of DVT
- It can measure the flow rate and detect regurge & turbulence of flow.
- Coloured duplex gives better interpretation because it shows the linear flow as red colour while the turbulent flow (e.g. regurge) appears blue.

4- Computed tomovenography (CTV) & Magnetic Resonance Veography (MRV):

- They are recent non-invasive investigations that have high accuracy in diagnosing proximal venous thrombosis but not in calf veins thrombosis, this with their high cost limit their use.

5- Radioactive (^{125}I) fibrinogen uptake: not used nowadays

Differential Diagnosis:

From other causes of tense tender calf :

1. Rupture plantaris tendon (tennis leg) or partial tear in the tendo-Achilis.
2. Post-traumatic calf haematoma
3. Deep abscess or cellulitis or erysipelas of the leg
4. Ruture Baker's cyst or gastrocnemius bursae

Treatment:

I- Prophylaxis:

A- Preoperative

1- Avoid preopertivebed rest

2- Mini heparin , 5000 U. 2 hours preoperatively, then 5000 U /8 hours till the patient is ambulant after the operation.

B- Operative:

1. Elevation of the patient legs, and avoidance of LL. compression during operation.
2. Intermittent calf compression or foot compression by pneumatic sleeves.
3. Galvanic current stimulation of the calf muscles during operation.

C- Post operative:

1. Elevation of the legs for 20 degrees with calf massage.
2. Elastic stocking (graduated) for the leg.
3. Early ambulation.

II- Treatment of established cases

I- Conservative Treatment:

A- Bed rest :

Bed rest and elevation of the leg to help venous flow until pain, oedema and calf tenderness disappear. This is done for at least 10 days which is the period needed for a thrombus to adhere to the vessel wall. Then elastic bandage is applied over the leg for 3-6 months

B- Anticoagulant therapy:

I- Heparin: should be the starting anticoagulant:

- *Action*: 1) It activates antithrombin III.
  2) It neutralizes factor IX, X & XI.
  3) It has a mild thrombolytic activity !!.
- *Dose* : - We usually start with 10.000 IU /6 hour by IV. infusion (better) or SC. route for adults then we adjust the dose according to partial thromboplastin time (PTT) which should be kept 2-3 times the normal
  - The heparin therapy should be continued till all local manifestations of thrombosis e.g. pain, tenderness, oedema disappear (usually 7-10 days).
- *Side effect* :- Bleeding tendency due to overdose. It is treated by IV protamine sulphate (heparin antidote) 1 mg /100 IU heparin.

NB. Fractionated heparin gives better results than non fractionated heparin. They have more prolonged action and lower incidence of bleeding

2. Oral anticoagulants: (Cumarin derivatives)

- *Action* : It blocks the synthesis of the four Vit K dependent clotting factors (prothrombin & factors VII, IX & X).
- *Dose* :-Start with 10mg warfarin then 5mg/day then adjust the dose according to the prothrombin time (PT) that should be kept 2-3 times the normal. Also, prothrombin concentration should be between 30% - 60% .
- *Side effect*: Bleeding tendency. Vit K is the antidote but should be combined with fresh blood transfusion or fresh frozen plasma (contain prothrombin).

- Oral anticoagulants are given during heparin therapy, which is stopped after 2-3 days.

- Oral anticoagulants should be continued for 3-6 months which is the period required for recanalization of the deep veins evidenced by duplex or ascending venography.

C) Thrombolytic therapy:

Fibrinolytic activators e.g. streptokinase, urokinase & tissue plasminogen activators (TPA).

- *Action* :They activate the fibrinolytic system and dissolve recent thrombi & may thus preserve the competence & function of the venous valves and limit the development of chronic venous insufficiency.

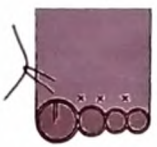
D) Novel Oral Anticoagulant: Rivaroxaban, Dibagatran, Apixban

II- Operative Treatment:

1- Venous thrombectomy with *Fogarty's venous catheter* .

Indications:

- Massive iliofemoral thrombosis to prevent venous gangrene.



Compartmentalization

2- I.V.C. (venous) interruption:

- *Indications:*

in pulmonary embolism to prevent recurrent embolism.

Mile's clip

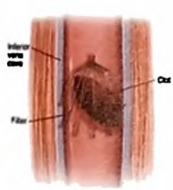
- *Methods:*

1- Clip application: Miles serrated clip around the IVC.

2- Compartmentalization of the inferior vena cava:

It means conversion of its single wide lumen into multiple small tracks by stitches to avoid the potential embolism.

3- Filter insertion in the I.V.C.: Greenfield filter (umbrella like filter) introduced through the internal jugular vein into the IVC. to filter dangerous emboli.



Greenfield filter

Pulmonary embolism

Aetiology:

1- Thrombo-embolism (the commonest): It complicates DVT.

2- Fat embolism: It may complicate bone fracture (femur), burns or liposuction.

3- Air embolism: may complicate major trauma, neck veins injury or. CV. Catheter.

4- Amniotic fluid embolism: It may complicate difficult labour.

5- Tumour embolism: It may complicate renal cell carcinoma.

Pathology:

-The embolism usually obstructs the lobar arteries in each lung.

-Pul. embolism and infarction are not synonymous as infarction occurs only in 10 % of cases.

- In fat embolism, the lipase enzyme in the lung will act on the fat globule liberating the free fatty which cause more damage to pulmonary circulation.

Complications: -

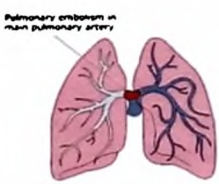
1- Death: in fatal type, DVT is the 3rd commonest cause of death.

2- Shock & cardiac arrest.

3- Pulmonary hypertension and RT side heart failure.

4- Low cardiac output, hypoxaemia & myocardial infarction.

5- Haemoptysis.



The fatal type

Incidence:

It is common after operations, pregnancy, recumbancy ..... etc

Clinical picture

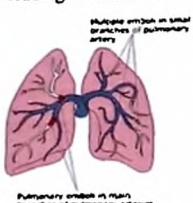
Manifestations usually appear in 7th to 10th postoperative day. The manifestations varies according to the size of the embolus:

1- Fatal type: with large embolus obstructing the main pulmonary trunk leading to sudden death.

2- Massive type: with obstruction of a main branch of pul. trunk : Severe chest pain , shock, dysnea & cyanosis leading to death in few hours.

3- Pulmonary infarction: with small embolus obstructing small branch :Chest pain , dysnea , cyanosis & may be haemoptysis.

4- Recurrent emboli: recurrent showers of minute emboli causing progressive obstruction of pul. capillary bed leading to pul. hypertension.



Investigations:

1- Blood gases: hypoxaemia is almost always present.

2-Serology: Elevated serum LDH , elevated S. bilirubin with normal AST is highly suggestive.

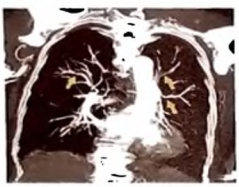
3- D diamer in the serum It is a fibrin specific degradation product results from endogenous fibrinolysis. It is more specific.

4- ECG: inverted T wave and depressed ST segment may be present

5- Chest X ray: Early there is no change.but later there is a typical wedge shaped opacity with its apex towards the hilum.

6- CT. pul. arteriography: the most diagnostic.

Chest X ray



CT. pul. angiography

Treatment:

I- Medical treatment:

1- Morphia to relieve pain.

2- O_2 inhalation may be with positive pressure ventilation.

3- Atropine to relieve bronchial spasm.

4- Antibiotics to guard against infection.

5- Anticoagulants: Heparin in full does should be started immediately.

6- Fibrinolytic drugs: streptokinase , urokinase & tissue plasminogen activator (TPA).They are injected through a catheter introduced through the jugular vein to the pulmonary artery.

* Na desoxylate may be used to dissolve fat emboli.

II- Surgical treatment:

A- Venous interruption: IVC interruption (see before).

B- Pulmonary embolectomy: (thrombolysis)

Indications:

Severe cases with persistent hypotension inspite of maximal resuscitation with proved massive pulmonary embolism by pulmonary arteriography.

Technique:

Thoracotomy and removal of the thrombus after cardiopulmonary bypass.

NB. Now, pulmonary embolectomy may be performed by less invasive method (transvenous pulmonary embolectomy) using *Greenfiled catheter* which is a large suction catheter introduced through the femoral vein up to pulmonary artery . It is more safe, less stressful to the patient and with better survival rate (up to 75 %).

Prognosis:

Mortality is more than 30 %.

Varicose Veins

Definition:

Dilatation, elongation & tortuousity of the veins

In lower limb. The varicosity affects only the superficial veins as they are not supported in the subcutaneous tissue while the deep veins are well supported by muscles & fascial septa.

Aetiology:

1- Primary varicose veins(1ry VV):

- They occur with no obvious cause and with patent comptent deep system. They occur due to:

A-Congenital incompetence of valves: leading to dilatation & tortuousity of the veins.

B- Congenital weakness of the wall of the veins: leading to dilatation of the vein and secondary incompetence of valves. This theory is supported by the common incidence of varicose veins with other mesodermal defects e.g. hernia, piles, varicocele & visceroptosis.

The predisposing factors:

-Prolonged standing or sitting, so, it is common in certain occupations e.g. surgeons, barbers.

2- Secondary varicose veins (2ry VV): due to:

A- Deep venous thrombosis & postphlebetic syndrome: (the commonest):

-DVT leads to obstruction of deep veins or incompetence of their valves after recanalization.

-There is venous hypertension & impaired muscular pump

B- Compression on deep veins:

-Pelvic, abdominal or femoral masses may compress deep veins and impair the bl. flow.

C- Arterio-Venous fistula (A-V fistula):

- Either congenital or acquired (traumatic) .

-It produces secondary varicose veins which are pulsating and don't collapse on lying down.

Pathology:

1- Primary varicose veins:

-The incompetence usually involves the valves of the communicators and the superficial veins.

-The deep veins are normal, so, the muscular pump is functioning and the venous drainage of the lower limb is not impaired.

- The varicosity affects the long & short saphenous systems.

2- Secondary varicose veins :

-The incompetence affects the valves of the deep veins and perforators.

-There is deficient muscular pump leading to impaired venous drainage of the lower limb.

-The varicosity affects any cutaneous vein, not mainly the greater or lesser saphenous vein.

Complications:

Complications of VV.

1-Thrombosis: thrombophelebitis.

2-Bleeding: from minor trauma . It is profuse bleeding *"the patient bleeds from the heart as there is no competent valves inbetween* ". It is controlled by elevation & pressure.

3-Pigmentations: Brownish pigmentations occur due to extravasation of RBCs from small venules with diffusion of haemosedrin granules into the tissues.

4-Pruritis: due to irritation by haemosedrin leading to itching.

5-Ulceration: due to

- *Theory 1: Fibrin cuff theory*
  - Extravasation of blood from small venules →haemosedrin deposition → irritation & scratching→ ulcer.
  - Also fibrinogen is changed into fibrin → fibrosis around capillaries → anoxia → ulcer .
- *Theory 2: leukocytes trapping theory*

  Leukocytes are trapped in the ulcer bearing area and release their mediators initiating destructive inflammatory process → ulcer .

- The ulcer has the following characters:
  - * *Site:* the ulcer bearing area above the medial malleolus in the lower 1/3 of the leg .
  - * *Size:* variable.
  - * *Shape:* early circular or oval. Late irregular.
  - * *Floor:* infected unhealthy granulation tissue.
  - * *Edge*: early: punched out. Late: sloping
  - * *Margin:* - wide pigmentations.
  - **Base:* mild induration. If it is over bone it may cause periostitis & localized bone thickening so, it becomes hard & fixed to bone & resistant to healing *(callus ulcer)*
  - **Discharge:* seropurulent discharge.
  - * *Inguinal LNs:* LNs are enlarged, firm & tender.

6- Oedema:

Oedema of the foot and ankle that increases at the end of the day .

N.B: All skin complications are more common & more grave in 2ry VV. than in 1ry VV .

Incidence:

Varicose veins are common in people with jobs that need prolonged standing e.g. barbers.

Clinical picture:

History: History of the cause in secondary VV. e.g. DVT or penetrating trauma .....etc.

Symptoms:

1-Pain:

-*Character* : dull aching. -*Severity* : mild in 1ry & severe in 2ry VV.

-*Site* : in the calf & foot. -*Radiation* : localized.

-*Increased by* : prolonged standing. -decreased by: elevation of the limb.

or sitting Walking in 1ry VV

2-Cosmetic disfigurement in females or failure in medical commission in males.

3-Ankle oedema: mild oedema in 1ry VV. and marked in 2ry VV.

4- Symptoms of complications: more in 2ry VV.

Varicose veins

Signs:

I- General examination:

1- Primary VV: Search for hernias, varicocele, piles, visceroptosis.

2- Secondary VV: Search for abdominopelvic mass.

II- Local examination:

-Varicose veins appear as bluish, tortuous, lax cystic, compressible swellings that disappear on elevation of the lower limb.

- *Saphena varix* is a saccular dilatation of the upper end of the greater saphenous vein due to incompetent saphenofemoral valve. It appears as a bluish lax cystic, compressible swelling in the upper part of the femoral triangle. It gives expansile impulse & thrill on cough .

-Varicose veins crossing the groin crease may be seen in case of obstructed or compressed iliofermoral vein (secondary VV.).

-VV due to A.V. fistula are pulsating with palpable thrill and audible continuous machinary murmur. The punch of veins persist after laying down. There is hyperdynamic circulation.

III- Special tests:

A-Tests to evaluate patency of the deep system.

1-Perthe's test:

-The patient lies supine & elevates the limb to evacuate the veins. firmly apply a crepe bandage from the toes to the groin to obliterate the superficial veins

-Ask the patient to do exercise for 15 minutes.

-If the deep system is occluded, patient will develop marked pain and can't continue.

- It is a subjective test depends on the patient's threshold for pain.

2- Modified Perthe's test :

- While the patient is standing, apply a venous tourniquet just below the saphenous opening to occlude the superficial veins.
- Ask the patient to do exercise for 5 minutes & observe the varicosites.
- If the deep veins are occluded, the varicose veins become more engorged with pain. If the deep veins are patent & competent, the veins will shrink or disappear (efficient pump). It is objective test.

Trendlenburg's test

B-Tests to detect the incompetent communicators.

1- Cough test: palpation of upper part of saphenous vein during cough will reveal a thrill when saphenofemoral junction is incompetent

2-Trendlenburg's test:

-While the patient is supine, elevate his limb to evacuate veins.

-Press with the thumb over the saphenous opening (1.5 inch below & lateral to pubic tubercle) or apply venous tourniquet just below it.

-Ask the patient to stand while pressing over the opening.

-Normally the greater saphenous vein fills slowly from below:

*If it fills rapidly from below ,this means incompetent communicators below the saphenofemoral junction.

*If it fills rapidly from above after removal of pressure, this means saphenofemoral incompetence (SFI) .

Multiple tourniquets test

3-Multiple tourniquets test:

-While the patient is supine & elevate his limb to evacuate veins.

-Apply four venous tourniquets to the thigh and upper leg.

-Ask the patient to stand and observe each segment of the vein between the tourniquets & sites of blow out are marked.

Two bandages test

4-Two bandages test:

-While the patient is supine, elevate his limb to evacuate veins.

-Apply crepe bandage from the toes to the groin.

-Ask the patient to stand & remove the bandage from above tie by tie while applying another bandage from above and observe the vein segment by segment for blowouts.

5-Manual localization "Two fingers test ":

-Exam. the VV. segment by segment through bidigital evacuation and

observe. if the segment inbetween fills → incompetent communicator.

6-Fegan method:

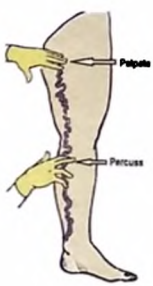
- Elevate the limb to evacuate the veins then palpate the course of the varicosites with a finger to detect gaps in the deep fascial (widened openings in the fascia that transmit the incompetent perforators).

Two fingers test

C-Test to detect incompetent superficial veins valves

Percussion test (Schwartz test):

While the patient is standing palpate the vein proximally & percuss a main bunch of varicosites distally. When the valves are incompetent, the percussion wave is transmitted to the palpating hand .It is a useful test to locate and mark the varicose veins in obesity or oedema.



Schwartz test

Investigations:

1-Ascending & desending venography

- Ascending venography to detect patency of the deep system and incomptence of communicators valves.
- Descending venography is performed by injecting the contrast into a catheter introduced into the femoral vein and ask the patient to strain to check for regurge and hence incomptence of deep system valves.

2-Doppler & duplex study:

Helpful in short saphenous disease and recurrent VV.

3-Arteriography:

To diagnose A-V. fistula in secondary varicose veins.

Differential Diagnosis

primary from secodary varicose veins

	Primary VV	Secondary VV
- History	-History of predisposing factors	-History of DVT, pelvic tumour,... etc
- Symptoms	-mild (pain, oedema...)	-severe (pain & oedema)
	- pain is improved by walking.	- pain is not improved by walking.
- Signs	-Bilaterality is common	-commonly unilateral
	-never cross the groin	-may cross the groin (iliofemoral DVT)
	-affect the saphenous system	-affect any subcutaneous vein
- Mod. Perth's test	-Decongestion of varicosites	-No change or more congestion
- Venography & Duplex	Patent competent deep system	-occluded or incompetent deep system

Treatment

I- Conservative ttt:-

Indications:

1-2ry varicose veins.

2-Mild 1ry VV with competent valves or spider varices.

Lines:

1-Avoid the predisposing factors in 1ry VV (prolonged standing or sitting).

2-Correct the cause in 2ry VV if possible e.g. pelvic tumours or A-V fistula.

3-Elastic stocking below knee to prevent the reflux and help the muscle pump.

4-Elevation of the foot of the bed to help venous drainage and avoid oedema.

II-Injection-compression sclerotherapy:

Indications:

1-VV. confined to below knee ,with competent saphenofemoral & saphenopopliteal valves.

2-Residual varicosites below knee after operation.

Materials:

1- sodium tetradecyle sulfate 3%. 2-sodium morrhuate 5%.

3-Ethanolamine oleate 5% . 4-sclermo or sclerovin (Polidocanol)

Aim:

to occlude the vein permanently so, injection should be done into an empty vein to cause intimal damage and permanent adhesion but not thrombosis, which otherwise may be dissolved with recanalization and recurrence.

Technique:

-Cannulate the vein while the patient is standing.

-Ask the patient to lie supine and elevate the limb to evacuate the vein.

-Inject small amount of the sclerosant material (0.5-1 ml) into the empty vein.

- Crepe bandage immediately to maintain the vein collapsed and help adhesions.

- encourge early walking to dilute the sclorsant material that would enter the deep veins.

- Repeat every 2-3 weeks till all varicosites are occluded.

Complications:

1-Permanent pigmentations at sites of injection.

2-Deep venous thrombosis if the material reached the deep system (sfi or spi).

3-Cellulitis & abscess formation when extravasation occurs.

4- Allergic reaction.

III- Surgical treatment:

Indications

1-Large varicose veins with incompetent valves.

2-Appearance of complications e.g. lipodermatosclerosis, bleeding ..etc..

Operations:

I-Ligation

1--Trendlenburg's operation : (saphenofemoral ligation)

Indications: Isolated saphenofemoral incompetence

Technique: Ligation of the greater saphenous vein in flush with the femoral vein with ligation of its main five tributaries (mention).

Triple ligation

2-Multiple ligation (Triple ligation):

Ligation of incompetent communicators and the involved segment then excision of the intervening segment .

II-Stripping:

*It is the most popular and effective method.

Technique:

1-The first step is Trendlenburg's operation.

2-The lower end of the greater saphenous is exposed in front of the medial malleolus and divided and the distal stump is ligated.

3-Introduce the stripper from the lower end of the vein and advance it till it appears from the upper end

4-Ligate the lower end of the vein over the stripper and pull the stripper firmly from above so, the bulky end of the stripper avulses the vein from below upward so the vein comes out crumpled on the stripper.

Stripping

5-Crepe bandage is applied while the limb is elevated to avoid bleeding from tributaries.

6-Allow walking from the second day. Crepe bandage is removed after one week.

N.B. Many surgeons prefere not to do stripping below knee to avoid injury of the saphenous & sural nerves that pass in close relation to the greater & lesser saphenous vein respectively.

III- Laser photocoagulation and Radiofrequency ablation:

These are new techniques done by special sets. A fine wire with active head armed with Laser or radiofrequency source is introduced in the saphenous veins as in stripping and during its slow withdrwal, it works to burn and coagulate the veins to oblitrate them

Venous (Gravitational) ulcer

Aetiology:

See before (mention in details)

Pathology:

Types: There are two types .

1- Varicose ulcer (rare): occurs in primary varicose veins .

2- Postphelibitic ulcer (common): occurs in secondary varicose veins

Venous ulcer

Characters: See before (mention in details).

Complications:

1- Malignant changes(Marjolin ulcer).

2- Callus ulcer, due to periosteitis (see before).

Clinical picture: (of postphlebitic syndrome):

1- Pain: that of chronic venous insufficiency.

2- Marked induration extends from the ankle up to the mid leg giving the leg the characteristic *"inverted champagne bottle "* or *" Turkey's leg"*.

inverted champagne

3-Ulcers & other complications e.g. pigmentation, & dermatitis.

Investigations:

I- Investigations to detect the cause: venography & duplex .

II- Investigations to detect complications: biopsy & X ray .

Inverted champagne bottle

Differential diagnosis:

From other ulcers of the leg (see later) .

Treatment:

I- Conservative ttt:

1- Elevation of the feet. 2- Daily dressing. 3- Passive & active exercise.

4- Below knee elastic bandage. .(4 layers compression technique) 5- Antibiotics.

II- Surgical ttt:

It is indicated in chronic cases with resistant ulcer.

1-Excision of the ulcer and covering the defect with Thiersch graft or flaps e.g. calf flaps.

2-Subfascial ligation of incompetent perforators (*Cockett & Dodd operation*). It is done through posterior median incision in the leg and attacking the perforators as they pass from muscle through holes in the deep fascia. This procedure can be performed with endoscopy through subcutaneous route i.e. subfascial endoscopic perforator surgery (SEPS).

The arteries

Acute ischaemia

Definition:

Sudden interruption of arterial blood supply to an organ or a limb.

Aetiology:

I-Traumatic:

1-Direct trauma: - penetrating trauma (stab, bullet) or blunt (crushing trauma).

2-Indirect trauma: neglected tourniquet or tight bandage or cast.

II Embolic:

An embolus obstructs the lumen. The source of emboli may be:

1-Lt. atrial thrombus on top of AF (commonest).

2-Vegetations on the mitral valve (endocarditis).

3-Mural thrombus on top of (Lt. ventricular infarction).

4-Detached atheromatous plaque on the aorta.

5-Detached thrombus from thoracic aortic aneurysm.

III Acute thrombosis:

It occurs due to the same causes of gradual thrombosis (Virchow's triad).

Pathology

I-Traumatic cases:

Site: usually at the site of wound.

Types: -

1-Closed injury (injury without division):

Arterial spasm or contusion may occur due to blunt crushing trauma or irritation by fractured ends of bone or by passage of a bullet near the vessel *(near miss injuries).*

2-Open injury (injury with division):

a) Complete division: Retraction and contraction of both ends will help haemostasis.

b) Incomplete division : Retraction and contraction of the vessel will enlarge the tear in the vessel wall, so, bleeding continues profusely.

II-Embolic cases:

Site: the commonest site for embolus impaction is the bifurcation of an artery e.g. bifurcation of the common femoral artery due to:

a) decrease in the diameter of the vessels b) turbulence of flow.

70% in the limbs 20 % in cerebral circulation 10 % in visceral arteries.

Sequelae:

Arterial embolism causes the following sequalae that worsen the distal ischaemia:

1-Reflex spasm of collaterals

2-Secondary thrombosis : occurs secondary to stasis & presence of embolus . It occurs:

a) proximally: propagating thrombus to the nearest major branch (collateral) ,and if not treated , thrombosis may extend to the collateral (bad prognosis).

b) distally: down the arterial tree to the tissues.

c) in the veins: DVT may complicate stasis.

3- possible further distal embolism.

III-Acute thrombosis:

-Site: The commonest site is the lower end of femoral artery.

- It Occurs in an artery already narrowed by atherosclerosis (acute on top of ch. ischaemia)

- Collaterals have been established before, so the degree of ischaemia is less than embolism.

Complications:

1-Gangrene: moist aseptic gangrene will follow if the circulation is not re-established rapidly and there is no sufficient collateral circulation.

2-Chronic ischaemia: if the condition is not adequately treated but the collateral circulation prevents the development of gangrene, the patient will pass into chronic ischaemia.

3-Deep veins thrombosis: due to stasis and venous spasm.

4-Compartmental syndrome: prolonged ischaemia causes oedema of muscles inside tight fascial compartment that may increase after reperfusion obstructing the circulation more & more and ischaemia may continue even after restoration of vascular patency.

5- Reperfusion injury (Post perfusion syndrome): During the period of ischaemia, the cellular and humoral elements activated by the hypoxia (complement, neutrophils, microvascular thrombi) are flushed back into the circulation where they cause further endothelial injury to organs such as the lungs and kidneys.

Clinical picture: -

Acute ischaemia is characterized by six **Ps** +1 **C**:

1-Pain: is sudden & characterized by:

-*Intensity* : severe. -*Character* : crampy & bursting .

-*Site* : begins at the site of obstruction and shoots along the limb distally.

-*Increased by*: exercise of walking -*Not relieved* by analgesics.

2-Paralysis: or paresis of the ischaemic muscles and loss of their tone.

3-Pufliness: oedema.

4-Pallor: which changes into mottled cyanosis due to accumulation of de-oxygenated blood .

5-Paraesthesia: progressive loss of sensation. Touch sensation is the earliest to be lost.

6-Pulselessness: loss of distal pulsation.(not in all cases)

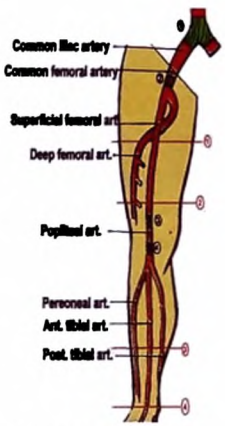
7-Coldness: the level of coldness (temperature change) depends on the level of obstruction:

a) Obstruction at aortic bifurcation → mid thigh level .

b) Obstruction of common femoral → just above knee level.

c) Obstruction of the upper popliteal→ lower leg level.

d) Obstruction of the lower popliteal → level of the maleoli .



The level of coldness

Investigations:

1-Doppler & Duplex study :to localize the site of obstruction.

2-Urgent angiography: to localize the site of obstruction and to assess the condition of collaterals.

Treatment:

I-Traumatic cases:

A-Fist Aid: Resuscitation.

B-Repair:

1-Injury without division : spasm & contusion:

a) Periarterial injection of papaverine or intra-arterial injection of heparinized saline.

c) Resection of the spastic or contused segment with venous graft .

2-Injury with division:

i) No gap : direct end to end anastomosis.

ii) Gap (1.5-2 cm) arterial reconstruction with graft either autogenous venous Graft for medium sized arteries or synthetic graft (Dacron, Gortex or teflon) for large sized ones.

II- Embolic cases:

A: Immediate full heparinization :

10.000 i.u. IV. bolus dose to prevent propagation of thrombus.

B-Definitive ttt :

1-Conservative ttt = fibrinolytic drugs

Streptokinase, urokinase, tissue plasminogen activator (TPA) may be used to dissolve emboli or thrombi .The results are not so good It is reserved only for cases unfit for surgery.

2-*Surgical ttt* = Arterial embolectomy:

a) *Immediate embolectomy*: Ideally, it should be done within 6 hs from the onset but can be done as long as the limb is still viable (present capillary circulation). Using Fogarty's balloon catheter under local anaesthesia, the embolus is extracted through small arteriotomy.

b) *Delayed embolectomy*: after 6-10 hours and may be performed even after irreversible tissue damage to lower the level of amputation. But, this procedure is associated with some serious complications e.g.

i) Crush syndrome: due to wash of mayoglobin from damaged muscles.

ii) Hyperkalaemia and cardiac arrest.

iii) Pulmonary embolism due to washing of thrombus from DVT to the lung circulation.

Embolectomy

III- Thrombotic cases

A-Immediate heparinization : as before.

B-Definitive ttt:

1- Fibrinolytic agents: as before

2- Surgery: thrombectomy or thrombendarterectomy or reconstructive arterial surgery (surgical bypass of occluded segment) after preoperative angiography.

N.B Fasciotomy may be needed after restoration of circulation to the limb to prevent compartemental syndrome.

-Long term anticoagulant or antiplatelet therapy is recommonded in embolic & thrombotic cases to prevent further emboli & thrombosis.

Chronic ischaemia

Definition:

It is chronic arterial insufficiency due to progressive gradual or intermittent arterial occlusion.

Aetiology

1-Atherosclerosis: (The commonest)

2-Arteritis :

-Thrombangitis obliterans (Burger's disease).

-Collagen diseases e.g. polyarteritis nodosa & scleroderma.

3-Vasospastic conditions:

- Raynaud's disease.

-Acrocyanosis.

4-External compression: e.g. Cervical rib.

5-Aneurysm: with thrombosis.

6-Acute ischaemia ending in chronic ischaemia.

Pathology:

Site:

-Atherosclerosis & Burger's disease commonly affect lower limbs.

-Cervical rib & Raynaund's disease commonly affect upper limbs.

Sequelae:

As arterial occlusion is gradual, collaterals have the time to develop. So, complete arterial occlusion may occur with minimal or even no symptoms. The tissues suffer from ischaemia more at stress & exercise. Trophic changes occur in longstanding cases.

Complications

1-Gangrene:

Dry aseptic gangrene occurs, as the vein is patent and there is enough time for evaporation of tissue fluid. It is precipitated by minor trauma or sepsis and commonly affects the big toe.

2- Trophic changes.

a) *Skin*: -dry scaly skin (loss of sweat & sebaceous gland) .

- Loss of hair.
- Loss of subcutaneous fat → Thin skin with tapering toes.
- Ischaemic indolent ulcers.

b) *Nails*: brittle and fissured.

c) *Muscles*: wasting with progressive weakness.

3- Intolerance to exercise: due to intermittent claudication & muscle weakness.

4- Insomnia: due to rest pain.

5- Impotence: in *Le riche syndrome*. (Aorto-iliac occlusion → bilat. LL. isch. & impotance).

Incidence:

Age:

- Atherosclerosis is common in old age.
- Diabetic atherosclerosis occurs in middle age.
- Cervical rib, Raynaud's disease and collagen diseases occur in young age.

Sex:

- Atherosclerosis & Burger's disease are common in males.
- Raynaud's disease, cervical rib & scleroderma are common in females.

Special habits:

- Burger's disease & atherosclerosis are commoner in smokers.

Clinical picture:

I- Symptoms:

1- Pain :

A) Intermittent claudication : This means ischaemic pain induced by walking or exercise that forces the patient to limp (claudication):

-*Intensity:* usually severe. -*Character:* crampy pain & sense of tightness.

-*Site:* depends on the site of obstruction i.e.

- Aorto-iliac disease causes gluteal claudication.
- Ileo-femoral disease causes thigh claudication.
- Femoro-popliteal disease causes calf claudication.
- Tibio-peroneal disease causes sole claudication.

- *Increased by :* exercise. -*Relieved by* : rest.

* *Claudication distance* is the distance, the patient walks before pain starts.
* *Claudication time* is the time, the patient walks on treadmill before pain starts.
* *Rest time* is the time, the patient rests before he can restart walking.

- As ischaemia progresses, the claudication distance & time are shortened while the rest time is prolonged

B)- Rest pain: It is due to nerve ischaemia and denotes advanced ischaemia:

-*Intensity:* severe. -*Character*: burning pain.

- *Increased by*: elevation, & warmth -Decreased by: dependency & uncovering

2- Paraesthesia: sense of numbness. Anaesthesia is present only in D.M.

3- Coldness: the patient may notice that one limb is cooler than the other.

4- Symptoms of complications e.g. trophic changes, ulceration, gangrene & impotence.

II-Signs

it is useful to use the dictum " Press and See How Colour Fades "

1- Pulse :

- The pulse is usually weak (stenosis) or absent (complete obstruction with).
- If pulse is felt, it disappears on exercise (disappearing pulse).

2- Sensation:

Diminished sensations but, complete sensory loss occurs only if there is associated neuropathy or impending gangrene.

3- Heat changes:

- The ischaemic limb is cooler than the other normal limb.
- The level of coldness depends on the level of arterial obstruction.

4- Colour changes:

i) In mild ischaemia, there is no colour changes.

ii) In moderate ischaemia, colour changes appear on elevation of the limb *(Burger's test)*.

Burger's angle: is the angle of elevation between the Limb and the horizon at which blanching occurs.

Burger's test & angle

It decreases with advancement of ischaemia. Burger's angle < 20 ° = impending gangrene.

iii) In severe ischaemia, there is fixed pallor even in the horizontal plane.

5- Functions:

There is wasting of muscles especially vastus medialis as it is a fleshy muscle.

6- Signs of complications:

Trophic changes (leg ulceration, hair loss, and dry scaly skin), and dry gangrene.

III-Special tests

1-Capillary filling time:

Elevate the limb till pallor occurs, then allow dependency and observe the return of colour to pink. If it takes more than 30 seconds, this denotes advanced ischaemia.

2- Harvey's venous refilling time:

Elevate the limb to right angle on the horizon till all veins empty then return it to the horizon and observe venous refilling. Normally, it takes about 10-15 seconds.

- If > 30 seconds = ischaemia
- If > 2 min = impending gangrene.

Fontain's clinical staging of chronic ischaemia:

- Stage I: Asymptomatic.
- Stage II: Intermittent Claudication.
  - Stage IIa: claudication distance > 200 meters.
  - Stage IIb: claudication distance < 200 meters.
- Stage III: Rest pain.
- Stage IV: Gangrene.

Signs of advanced ischaemia = warning signs of impending gangrene

1- Rest pain
2- loss of sensations (without neuropathy)
3- Burger's angle < 20°
4- Ankle-brachial index < 0.3 (see below)
5- Capillary filling time > 30 sec. 6- Venous efilling time > 2 min.

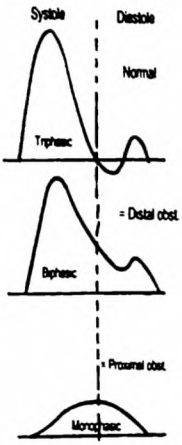
Investigations :

1-Plain X ray:

It may reveal calcified wall of the artery in rare cases.

2- Doppler U/S:

1) It gives an idea about the degree of ischaemia & site of obstruction:
   - Normally over medium sized arteries, it is triphasic.
   - Proximal to obstruction, it is biphasic.
   - Distal to obstruction, it is monophasic with low amplitude.
2) Useful in measurement of systolic bl. pressure at the ankle and measurement of Ankle-Brachial Index (ABI) which is the ratio between systolic bl. pressure at the ankle and that at the arm. Normally, it is > 1. If it is below 0.8, it indicates ischaemia. If it is less than 0.3 ,it denotes advanced ischaemia with rest pain



Duplex U/S

3- Duplex U/S:

- It combines imaging of the vessel and flow study.
- It gives quantitative data about the velocity of flow, site of obstruction, degree of narrowing plus the condition of collaterals and the vessels below obstruction.

5- Angiography:

Radiological exam. of the arterial tree after injection of a contrast.

Value of angiography:

-It detects the site & extent of obstruction.

-It detects extent of collateral circulation.

-It detects arterial flow distal to obstruction (*distal run-off*).

Types:

1) Transfemoral angiography: under local anaesthesia, a catheter is passed through the femoral artery up to the aorta. Angiography can be performed on any part of the body selectively.

2) Translumbar angiography: (not done nowadays)

When both femoral arteries are occluded, the catheter is introduced from the back under U/S guide. It may cause large retroperitoneal haematoma

Angiography

3) Transbrachial angiography:

The catheter is passed through the brachial artery, when both femoral arteries are occluded.

4) Digital subtraction angiography (DSA) :

The dye is injected intravenously. The dye will reach the heart and then to the arteries. Serial X-ray films are taken before & after injection of the dye. By computerized subtraction of images, a clear arteriogram is obtained. It is less invasive method .

5) Comupted Tomo-Angiography (CTA) & Magnetic Resonance Angiography (MRA): They need no contrast and give very valuable data.

6) Intra–opertive angiography:

Direct cannulation of the artery during operation.

5- Venous occlusion plethysmography (plethysmos= enlargement): Historical

Occlusion of venous return by inflating a cuff around the thigh to 50 mmHg then recording the change in volume of the limb with each heart beat which is proportional to the arterial flow.

6- Medical sympathectomy :

Injection of local anaesthetic around the sympathetic chain (paravertebral block) to:

1. determine the presence of spasm.
2. evaluate the benefit of sympathectomy.

Treatment:

I- Conservative ttt: -

Indications:

1- Mild cases (stage I & stage IIa)

2- Unfitness for surgery and recent coronary or cerebral thrombosis

Lines:

A- General measures

1-Improve general condition, correct anaemia , & control of D.M.

2- Prohibit smoking and avoid alcohol.

3-Drugs a- Papaverine I.M .injection (vasodilator) (ineffective in atherosclerosis).

b- Pentoxifylline (Trental) increases RBCs flexibility .

c- Prostavasin (PGE1) : vasodilator , & increases RBCs flexibility.

d- Platelets aggregation inhibitors : Aspirin in low dose is very effective.

B- Local measures:

1-*Burger's exercise:* elevation of the limb for 2 minutes and dependency for 2 minutes repeatedly till pain occurs.

2-Repeated washing, cleansing, drying & powdering of the diseased limb.

3-Cautious cutting of nails & callosities.

II- Surgical ttt:

A- Arterial reconstructive surgery

Indications:

1- Rest pain and severe claudication pain (stage IIb, III)

2- Presence of distal run off is a must for feasibility of arterial surgery.

Procedures:

1) Thrombendarterectomy :

- Removal of the thrombus together with the diseased intima through either long arteriotomy or using endarterectomy loop stripper.
- It is commonly used for carotid atherosclerosis.

2- Arterioplasty: e.g. profundoplasty

-Dilation of stenosis by different techniques including venous patch insertion.

-Profundoplasty means dilatation of the orifice of the profunda femoris with venous patch

N.B The profunda femoris artery itself usually escapes atherosclerosis due to:

1- its curved course protects it from the force of bl. stream.

2- deep among muscles & away from joints (not liable to trauma).

3- Arterial bypass operations:

a) Anatomical bypass operations:

- the occluded segment is bypassed from above to just below the obstruction

Examples:

*Femoro-popliteal bypass and femoro-posterior tibial bypass using either reversed saphenous graft (to change the position of valves) ,or better in situ saphenous bypass after destruction of valves with a valvutome.

Femoro-popliteal bypass

In situ bypass is better because:

i. Long bypass can be performed without kink or twist.
ii. Intact vasa vasorum (better nutrition).
iii. Blood stream is getting faster as the lumen narrows.

* Aorto-bifemoral bypass, using synthetic arterial graft .

Aorto-bifemoral bypass

b- Extra-anatomical bypass operations

Examples:

* Axillo-femoral bypass or axillo-bifemoral bypass.

* femoro-femoral cross over graft using synthetic arterial graft.

Types of arterial grafts

1- venous graft, e.g. saphenous vein (the most popular)

2- Arterial graft: e.g. radial art . (the best)

3- Artificial graft: Dacron , teflon , Gortex.

Axillo-femoral bypass

femoro-femoral cross over graft

B- Sympathectomy: (not done nowadays)

Indications:

1) Chronic ischaemia with no distal run off .

2) In conjunction with direct arterial surgery to improve results.

3) In conjunction with amputation to promote healing of wound .

Technique:

* Cervical sympathectomy for upper limb ischaemia (denervate cervical ganglia). It may be performed through throracoscope with lower morbidity.

*Lumber sympathectomy for lower limb ischaemia. It denervates 2nd & 3rd lumbar ganglia. The first lumbar ganglion may be removed only unilaterally to preserve ejaculation.

N.B. The claudication pain usually worsens after sympathectomy (↓↓ bl. supply to muscle).

C- Amputation

Indications:

1- Spreading gangrene endangering patient life.

2-Intractable infection with toxaemia.

3- Severe pain (rest pain) causing insomnia and disturbing the patient's life.

Technique:

-Different levels of amputation according to the site of obstruction.

*Above knee amputation is the usual level but, below knee amputation may be used if popliteal pulsation is felt . Amputation may be combined with sympathectomy to allow healing of the wound.

III- Interventional treatment:

A- Balloon transluminal dilatation.

B- Percutaneous transluminal angioplasty (PTA): Splitting or destruction of the atheroma (may be with laser beam or a revolving instrument) then balloon dilatation.

C- Application of stent: after angioplasty to avoid restenosis.

Percutaneous transluminal angioplasty

Atherosclerosis

Definition

Degenerative arterial disease characterized by atheroma formation & irregular lumen narrowing.

Aetiology

It is an aging process of the arteries predisposed to by:

-DM , hypertension , smoking , alcohol , hypercholestrolaemia.

Pathology:

Site :- Atherosclerosis affects large & medium sized arteries.

* Large sized arteries: Aorta and its main branches.

* Medium sized arteries: -Coronary & cerebral (common).

- Lower limb arteries (next common).

-There is subintimal deposition of lipid material forming a plaque called " atheroma" which causes irregular narrowing of the artery with chronic ischaemia.

Monkeberg's median sclerosis is a distinctive form of atherosclerosis affecting the fermoral arteries and characterized by sclerosis and calcification of the media.

Clinical picture, investigations & treatment:

See chronic ischaemia

Buerger's disease

(Thrombangitis obliterans)

Definition:

Disease of unsure aetiology characterized by inflammation and thrombosis of small neurovascular bundles of the limbs.

Aetiology : (unsure)

It may be due to hypersensitivity to tobacco as all patients are smokers .

Pathology :

Site: It is pachy in distribution.

- It affects distal arteries of the limbs especially lower limb below the popliteal trifurcation.

-It begins in the lower limb then affects the upper limb and less frequently affects visceral arteries (renal, mesentric, coronary and cerebral).

-There is inflammatory reaction that entangles the artery, the vein and the nerve with dense fibrosis , thrombosis and oblitration of lumen .

-Calcification never occurs .

Complications :

As in chronic ischaemia, but gangrene and rest pain are earlier.

Incidence :

It occurs in young adults 20-40 y male smokers (usually heavy smoker) more common in jews.

Clinical picture :

I- Prodroma :

Thrombophlebitis mirgans (Trauseau's sign) and Raynaud's phenomenon are common .

II- Picture of chronic ischaemia

1- Claudication pain in the sole .

2- Rest pain is early and severe (the nerve is included in the inflammatory process) .

3- Gangrene in the toes (or fingers).

Investigations : See ch. ischaemia

.Angiogrphy shows arterial occlusion with no distal run off giving ***the tree root appearance.***

Differential diagnosis: From other causes of ch. Ischaemia especially atherosclerosis.

	Atherosclerosis	Burger's disease
Patient	Old age more, in male smokers	Young adults only in smoker males
Site	Cerberal, coronary, aotoiliac, femoropopliteal.	Distal vessels and patchy in distribution
Pathology	- The intima & internal elastic lamina.	- Internal elastic lamina is free
	-The veins & nerves are free	- The veins & nerves are affected
	- Calcifications may occur	- Calcification never occurs
Rest pain	Rare & late	- common and early
Associations.	Raynaud's phenomenon	- Raynaud's & migrating thrombophleb.
Angiography	- Possible distal run-off	- no distal run off (tree root appearance)

Treatment:

I- Conservative ttt : (as before)

Stop smoking *"Either your limb or your cigarettes"*

II- Surgery:

- Direct arterial surgery is not feasible (no distal run off).
- Sympathectomy may be beneficial.
- Amputation : indicated in :

  a- gangrene of the toes , conservative amputation for dry gangrene .

  b- severe persistent rest pain .

  c- urgent amputation (below knee) for spreading gangrene .

Raynaud's disease

Definition :

A vasospastic disease affecting small arteries & arterioles in response to coldness.

Aetiology :

1- Sympathetic overactivity (not accepted) .

2- Hypersensitivity to coldness .

3- Psychological instability .

Pathology :

Site: -It is bilateral and symmetrical.

-It affects small arteries & arterioles of the extremities.

-It affects the upper limbs & less commonly lower limbs.

Pathological changes: In response to cold or emotions, there are three consecutives phases :

1- Pallor: due to spasm of digital arteries .

2- Cyanosis : due to dilatation of capllaries which are filled with cyanosed deoxygenated blood .

3- Redness: when the attack is off , reactive hyperaemia will follow and the capillaries will be flushed with oxygenated blood.

Complications:

1-Thrombosis of digital arteries. 2- Trophic changes & gangrene of the finger tips.

Incidence:

It is more common in young adult females .

Clinical picture :

1-The attacks are precipitated by cold or emotional excitement. There is typical colour changes (pallor, cyanosis then redness). Pain may be severe during the attack and subsides gradually.

2- Picture of complications , e.g. gangrene of finger tips.

Investigations:

Cold test: Immersion of the patient's hands in cold water may precipitate the attack .

Differential Diagnosis:

From *Raynaud's phenomenon:* The same colour changes may occur in conjunction with other conditions or organic diseases such as:

a) Occupation: typists, pianists & using vibrating tools.

b) Vascular diseases: atherosclerosis, Burger's disease & scalene syndrome.

- It is mostly unilateral, affecting feet more than hands ,and trophic changes are rare.

Treatment:

I- Conservative measures: for mild cases:

1- Avoid exposure to cold & wearing wool gloves in cold weather.

2- Small doses of thyroxine (5μg tds) to cause VD & Reserpine (sympatholytic) .

3- Severe persistent attacks should be treated by intra-arterial injection of reserpine 1mg in brachial artery + heparinization .

II Surgical ttt: for severe cases.

Cervical sympathectomy better through thoracoscope. It gives good results.

Scalene syndrome

(Thoracic outlet syndrome - Cervical rib)

Definition:

It is a neuro-vascular syndrome due to compression of the trunks of brachial plexus and subclavian artery at the thoracic outlet (the triangle formed by scalenus anterior muscle, anteriorly, scalenus medius muscle posteriorly & the 1st rib inferiorly).

Aetiology:

I- Causes:

1- Presence of cervical rib:

-An extra rib that originates from the transverse process of the 7th cervical vertebra.

-It may be complete or incomplete with tapering end that is usually connected by a fibrous band to the 1st rib, or may be completely unossified and represented by a fibrous band that extends from cervical vertebra to the 1st rib.

2- Muscular abnormality:

a - Abnormal attachment of scalene muscles that leave small space between them.

b- Tight scalene muscles.

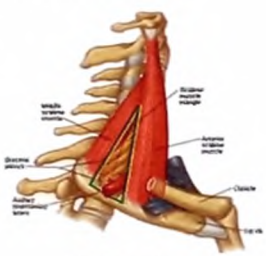
II- Predisposing factors:

Neurovascular compression is precipitated by dropping of the shoulders which may be due to :

1- Rapid growth during puberty.

2- Muscle weakness.

3- Lifting heavy weights on the shoulder.



Scalene triangle

Pathology:

Site: It is more common on the right side (dominant arm) .

Pathological changes: Compression of the brachial plexus & subclavian art. occurs as they pass through the thoracic outlet. The subclavian vein passes in front of the scalenus anterior muscles outside the triangle, so it escapes compression.

Complications:

1-Neurological complications : Both sensory, motor & autonomic effects.

2-Chronic ischaemia: due to compression (stenosis) of subclavian artery.

3-Poststenotic aneurysm: due to maldirected flow of bl. causing aneurysmal dilation that may harbor a thrombus with possible embolism that usually affects the digital artery of the index.

Incidence:

It is more common in adolescents and young adult females.

Clinical picture:

Symptoms:

I- Neurological manifestations: (more common) due to compression on C_8-T_1.roots.

1- Sensory: shooting pain along the medial side of the upper limb with numbness over the ulnar side of the hand.

2- Motor: weakness and atrophy of the small muscles of the hand.

3- Autonomic: stimulation of sympathetic fibres in T_1 may cause Raynaud's phenomenon.

II Vascular manifestations:

1- Intermittent ischaemic pain on exercise relieved by rest.

2- Paraesthesia , coldness & pallor.

Signs:

1- Unequal radial pulsation.

2- Palpable cervical rib (rarely).

3-Palpable thrill or audible bruit over the distal part of subclavian artery.

Special test:

Adson's test: while the upper limb is hanged down palpate for radial pulsation then ask the patient to turn his head to the opposite side., the pulse will disappear or become weaker.

Investigations:

1-Plain X ray: Radiological findings don't usually correlate with severity of the disease as many cases with large cervical rib are asymptomatic while others with free X ray films have severe symptoms. MRI is of great help.

Treatment:

I- Conservative ttt (for mild cases):

Physiotherapy to muscles of the shoulder to elevate it.

II- Surgical ttt: (for severe cases):

a- If there is cervical rib: Excision of the cervical rib within its periosteum may be with scalenotomy (division of scalenus anterior muscle).

b- If there is no cervical rib :

1- Scalenotomy. 2- Transaxillary resection of the first rib.

Aneurysm (أم الدم الشريانية)

Definition:

Localized dilatation of an artery, Or sac filled with blood or thrombus connected to an artery.

Aetiology:

I- Congenital: congenital aneurysms in circle of Willis, renal or splenic artery.

II- Traumatic: trauma may cause aneurysm through:

1- Traumatic contusionwhich causes saccular aneurysm.

2- Haematoma formation that maintains its communication with the artery (False aneurysm).

III- Inflammatory:

1- Syphilis: It causes aneurysm of the aortic arch.

2-Mycotic aneurysm: secondary to infective endocarditis.

IV- Degenerative:

Atherosclerosis (elasticity of the vessel protects it from trauma of the blood stream).

N.B The commonest causes are atherosclerosis and traumatic contusion.

Pathology:

Site

	Aortic	Femoral	Popliteal
Incidence:	Common	Rare	Common
Aetiology:	Atherosclerosis	Atherosclerosis	Atherosclerosis
	syphilis	trauma	trauma
Complications:			
Distal ischaemia	Rare	Common	Common
Rupture	Common	Rare	Frequent

Types:

1- Fusiform aneurysm: (The commonest) It is usually due to atherosclerosis & may be syphilis.

2- Saccular aneurysm: such as traumatic , mycotic & congenital aneurysms.

3-Dissecting aneurysm: It is usually secondary to atherosclerosis & hypertension. There is a crack in the intima and blood dissects between the inner & outer layers of the media. Dissection may extend distally to the femoral arteries and proximally to the aortic valve.

Complications:

1- Thrombosis & distal embolism.

2- Distal ischaemia: due to narrowing by thrombosis.

3- Calcification.

4- Inflammation: infection of its thrombus with possible pyaemia.

5- Rupture: two types:

- Slow rupture (leaking aneurysm) with marked pain .
- Rapid fatal rupture.

6- Compression on the surrounding structures:

- *Artery:* causing chronic ischaemia.
- *Veins:* congestion, oedema & secondary varicose vein .
- *Nerves:* causing hypostheisa, paresis.
- *Viscera:* oesophagus → dysphagia trachea → dypsnea ....etc .
- *Bone:* causing bone resorption & may be pathological fracture e.g. vertebral bodies destruction in aortic aneurysm. Interverterbal discs are least affected, as they are avascular.
- *Joint:* popliteal artery aneurysm produces destruction of knee joint .

Saccular aneurysm

Fusiform aneurysm

dissecting aneurysm

Incidence:

Atherosclerotic aneurysm is common in old age while traumatic one is frequent in young age

Clinical picture:

I- Mass: which is characterized by

1- Rounded or oblong smooth cystic mass in the course of an artery.

2- Mobile across but not along its artery (unless there are adhesions).

3- Compressible, pulsating (systolic & expansile) with systolic thrill & murmur (bruit).

4- Proximal compression decreases the aneurysm size with loss of its pulsation, thrill & murmur. Release of pressure makes it regain its size & pulsation after 2-3 heart beats.

5- Distal compression leads to increase in the size of the aneurysm (risky).

II – Picture of complications:

Such as ischaemia , rupture or inflamation , Secondary VV., bone erosion.

Investigations:

1- US, MRA & CTA: investigations of choice especially in aortic aneurysm.

2- Angiography: It may not show the aneurysm due to presence of intraluminal thrombus, but it shows the condition of the artery below and above the aneurysm and state of collaterals.

Differential Diagnosis:

From other pulsating masses:

Two fingers test

1- A-V- aneurysm (fistula) :

1- The murmur is continuous machinery.

2. Thrill & murmurs propagate both proximally & distally.

3. Secondary varicose veins are always present and are pulsating.

2. Highly vascular tumours (sarcomas):

1- The pulsations are wavy (shaking). Each arteriole pulsates separately.

2- Proximal compression does not decrease the size of the mass.

3- Masses over on artery (with transmitted pulsation):

1- When the mass is brought away from the artery (knee-elbow position in pancreatic pseudocyst), the pulsation disappears.

2-Two fingers test: the fingers are only elevated (not separated) with each pulsation.

3-Proximal compression doesn't alter the size but stops the pulsation which return immediately after release.

Treatment:

I- Aneurysm of an unimportant artery.

A- Ligation- excision: is the best.

B- Ligation alone :

1- Proximal & distal ligation is the best.

2- Proximal ligation :.

3- Distal ligation: in case of thrombosed aneurysm for the fear of thrombo-embolism.

N.B. Ligation of the aneurysm is no more used and replaced by the more recent arterial reconstructive technique. Ligation operations are preserved for inflammed aneurysm .

II- Aneurysm of important arteries:

A- Excision and graft replacement: the best.

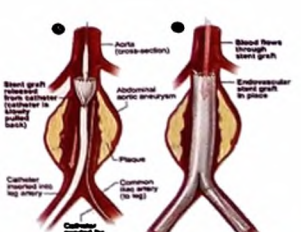
B- Exclusion and graft bypass:

C- endovascular aneurysm repair (EVAR):

Now, if excision is risky, the aneurysm can be repaired through endovascular (endo-luminal) stent graft application e.g. infrarenal aortic aneurysm

D- Aneurysmorrhaphy:

Not done nowadays



endovascular aneurysm repair

III–Amputation is indicated in:

1) Gangrene.

2) Infected aneurysm.

3) Disorganized or paralyzed limb.

IV–False aneurysm:

Evacuation of the haematoma and repair of the artery may be with venous patch.

Arterio-venous fistula or aneurysm

Definition:

Abnormal communication between artery & vein.

Aetiology:

I-Congenital: as in Robertson's giant limb (localized & diffuse forms) .

II-Traumatic (the commonest):

A- Accidental: due to transfixing injury of both artery & vein e.g. stabs, bullets.

B- Artificial: therapeutic A-V. fistula for haemodialysis in renal failure.

C- Iatrogenic: due to transfixed ligation of vascular pedicle of organs containing both the artery & vein.

III- Pathological: A-V fistula in thyrotoxicosis.

Pathology:

The blood shifts directly from the artery to the vein bypassing the tissues causing:

1- chronic ischaemia of the limb.

2- hyperdynamic circulation with tachycardia.

3- secondary varicose veins with venous hypertension.

Complications:

I-General:

Lt. ventricular hypertrophy and high output failure.

II- Local:

1-Chronic ischaemia with all its complications.

2-Secondary varicose veins with all its complications.

Clinical picture:

- History: of trauma or operation.

- *General:*

- Tachycardia & collapsing pulse (Hyperdynamic circulation).

- *Branham's bradycardiac reaction:* slowing of the heart rate on compression of the artery or the fistula.

A-V fistula

- Local manifestations:

A-Mass: A-V aneurysm has all characters of arterial aneurysm with the following differences:

1- The murmur is continuous machinery.

2-The thrill & murmur propagate both proximally & distally.

3- Secondary varicose veins are always present and pulsating.

B- Manifestations of complications: e.g. 2ry VV., local gigantism

Repair of both art. & V.

Investigations:

Angiography: Appearance of the veins in the arterial phase

Treatment:

I- Important artery & vein.

Excision & repair of both artery & vein.

Repair the art. alone

II- Important artery & unimportant vein:

Repair of the artery on the expense of vein.

III- Unimportant artery & vein:

Quadriple ligation: ligating both the artery & vein, both above &below the fistula.

Quadriple ligation

Gangrene

Definition: -

Death of macroscopic piece of tissue usually with putrefaction.

Aetiology

I- Vascular causes :

1-Acute ischaemia: causes wet aseptic gangrene.

2- Chronic ischaemia: causes dry aseptic gangrene.

3- Venous gangrene: are due to massive iliofemoral thrombosis.

II- Trauma:

1-Direct trauma: crushing of the tissues, prolonged pressure by cast or the bed (pressure sores).

2- Indirect trauma: through vascular injury.

III- Infection: (infective gangrene): e.g. Diabetic infective gangrene (the commonest), Gas gangrene, *Fournier's gangrene* of the scrotum, & carbuncles.

Pathology:

Types:

1- Dry aseptic gangrene:

Dry gangrene

-It occurs in chronic ischaemia as there is enough time for evaporation of tissue fluid and the venous system is patent.

-The gangrenous part becomes shrunken, dry, wrinkled with greasy surface (mummified) with black discoloration due to diffusion of disintegrated blood pigments.

2- Moist aseptic gangrene:

Moist gangrene

- It occurs in acute ischaemia as there is no enough time for evaporation of tissue fluid and associated venous spasm causes oedema.
- The gangrenous part is usually of the same size & consistency but becomes discolored gradually to blackish green discoloration with skin blebs & offensive odour.

3- Moist septic gangrene:

-The gangrenous part becomes swollen, oedematous with multiple bullae filled with highly offensive infected serum with marked toxaemia.

Sequelae:

The gangrenous part usually separates by the following mechanisms:

1- Line of demarcation: when gangrene reaches part with good bl. supply, it stops and the viable tissue reacts against it with inflammatory reaction forming a zone of hyperaemia called "*line of demarcation* " .

2- Line of separation: ulcers begin to appear at the junction with the gangrenous tissues, which fuse with each other to form "*a line of separation* ".

3- Casting off: the line of separation deepens gradually till the gangrenous part separates. As the vascularity of the deep structures (muscles, bone.... etc) is better than that of the skin & S.C. tissue, so gangrene separates leaving a conical stump.

Clinical picture:

The cardinal signs of gangrene follows the dictum *"Press and See How Colour Fades"*

1- loss of **P**ulsation. 2- loss of **S**ensation. 3- loss of **H**eat.

4- **C**olour changes. 5- loss of **F**unction (movement).

6- loss of capillary circulation (when you press, no colour fades). If the capillary circulation is still present, it is imminent (threatened) gangrene. If absent, it is established gangrene.

Diabetic gangrene

There are two major types of diabetic gangrene:

I- Diabetic atherosclerotic gangrene

(Diabetic macroangiopathy)

- Diabetic patients are more prone to atherosclerotic changes at earlier age group.
- Thrombosis & obstruction affect major vessels of the limb.
- There is no distal pulsation.
- The gangrene begins dry but commonly becomes infected.

Clinical picture, Investigations & treatment as in chronic ischaemia.

N.B. Sympathectomy is useless in diabetics as diabetic neuropathy acts as auto-sympathectomy.

II- Diabetic infective gangrene

(Diabetic microangiopathy)

See foot surgery

Decubitus ulcer

Pressure (Bed) sores

Aetiology :

Prolonged pressure on skin against bone interferes with tissue perfusion →traumatic gangrene.

Precipitating factors:

1- pressure of the bed over bony prominences .

2- pressure of plaster cast or Thomas splint over the skin .

Pathology:

Site: Over bony prominence e.g. sacrum, ischial tuberosity, greater trochanter, heels & scapular blades.

Pressure sores

Treatment:

-Repeated dressing then reconstruction with local flaps.

Gangrene of the big toe (Blue Toe Syndrome)

The commonest causes are:

1- Diabetic infective gangrene

2- Atherosclerotic (ischaemic) gangrene

3- Burger's disease and other types of arteritis

4- Embolism by detached thrombus or atheromatous plaque from proximal diseased artery or aneurysm (big toe syndrome)

Lymphatic system

Lymphoedema

Definition:

Definition: Lymphedema is a gradual limb swelling which results from excessive accumulation of extracellular fluid in the interstitial compartment due to defective lymphatic function

N.B. Elephantiasis is a descriptive term that describes a hypertrophic condition of skin & S.C. tissue simulating those of an elephant.

It is caused by different diseases e.g:

1- Chronic lymphoedema 2- Ch. venous (postphlebitic) oedema .

3- Bilharzial pseudo-elephantiasis. 4- Elephantiasis neuromatosa.

5- Congenital A-V fistula.

So, elephantiasis is not a synonymous for lymphoedema as usually thought.

Aetiology:

I- Congenital (Primary) lymphoedema:.

-It is due to hypoplasia of cutaneous lymphatics e.g. *Milroy's oedema*.

-It is more common in females.

-It may manifest at birth *(lymphoedema congenita)*, at adolescence *(lymphoedema precox)* or at later adult life *(lymphoedema tarda)*.

II- Acquired (secondary) lymphoedema:

A- Trauma:

1- Circumferential trauma: circumferential burn or crushing injury obstructing the lymphatics all around.

2- After block dissection of L.N.s. e.g. axillary or inguinal block dissection.

B- Infection:

1- Specific infection: e.g.. T.B lymphadenitis, & erysipelas.

2- Non specific infection: e.g. recurrent streptococcal lymphangitis.

3- Parasitic infestation: e.g. filariasis (the commonest cause in tropics).

C- Neoplasms:

Heavy malignant metastases in LNs obstructing lymphatics e.g:

1- lymphoedema of the UL due to secondaries in axillary LNs in cancer breast.

2- Lymphoedema of the LL due to secondaries in inguinal LNs in cancer scrotum & anal canal.

Flariasis is the commonest cause of lymphoedema in tropic areas:

-It is caused by the nematode worm *(Wuchereia Bancrofti)*.

-The adult worms live in the lymphatics & cortex of human lymph nodes.

-They lay microflaria which reach the circulation through thoracic duct & circulate with blood especially at night (nocturnal periodicity).

- Microflaria are sucked by the intermediate host *(Culex Pipiens)* mosquitoes where they mature into embryo flaria which are the infective stage to human.

- Embryoflaria are injected into human body through mosquitos' bite where they reach the lymphatic system and mature into adult.

Pathology:

Site: - It commonly affects L.L., scrotum, UL, breast in this order of frequency.

Stages: It passes into the following stages:

1- Stage of pitting oedema:

It is an early stage. Lymphatic vesicles may appear and rupture discharging their fluid (lymphorrhea).

2- Stage of non pitting oedema:

Lymph fluid is rich in protein which is irritant to the tissues, stimulating their growth & initiates a fibrotic process in skin & S.C. tissues. So, oedema becomes non pitting.

3- Stage of pseudopapillae formation:

- They are warty growths which usually appear on the dorsum of the foot.
- The skin appears rough, puckered & rugose, like that of an elephant.

N.B -lymphoedema doesn't affect the tissues deep to the deep fascia as they are rich in lymphatics and the muscle contraction helps lymphatic flow and prevents stasis.

Complications:

1- Recurrent lymphangitis.

2- lymphoedema ulcer (ruptured lymphatic vesicles).

3- Heavy bad sighted limb may interfere with body activity.

4- Marked lymphoedema of the scrotum may cause mechanical impotence (sunken penis).

5- lymphangiosarcoma (rare) may complicate long-standing cases.

Incidence:

-It is endemic in certain districts where the mosquitoes habitant is prevalent e.g. Sharkia, Rasheed, Mansoura & Giza.

Clinical picture:

I- History: of the cause:

- History of recurrent lymphangitis with fever (elphantoid fever).
- History of trauma or operation.

II-Lymphoedema : with its three stages

1- Pitting oedema : more at the end of the day.

2-Non pitting oedema: with thick skin & SC. tissue ,but oedema is absent in:

a) skin around the ankle joint.

b) sole of the foot.

Lymphoedema LL.

This is because these areas have poor SC tissues and their lymphatics drain directly into lymphatic of the joint below. Pressure on the sole during walking is an additional factor.

Stemmer sign: inability to pinch the skin over second toe.

3- Warty pseudopapillomatosis.

Investigations:

I- Investigations to prove flarial aetiology: Blood film at midnight to detect microflaria.

II- Lymphography:.It has two methods:

1-Visual method: through injection of patent blue dye in the web space between the toes to delineate lymphatic vessels of the lower limb.

2- Radiological method: Visual method is done first, then a suitable lymphatic vessels is canulated through incision on the dorsum of the foot and a radioopaque material (ultrafluid lipidol) is then injected slowly with a special pump to delineate lymphatic vessels

III- Lymphoscintigram: It is now the golden standard for assessing the lymphatics. The radiolabeled protein used is Tc 99m–labeled colloid, It allows measurement of lymphatic function, lymph movement, lymph drainage, and response to treatment.

Different Diagnosis:

1- Unilateral cases

From other causes of elephantiasis (see before)

2- Bilateral cases:

1- From other causes of generalized oedema:

a- Cardiac b- Hepatic c - Renal d- Nutritional

e- Endocrinal (Cushing's disease – contraceptive pills).

2- Causes of unilateral oedema when occur bilateral

Treatment:

I- Conservative ttt: in early mild cases

-Rest with elevation of the leg. -Elastic Stocking. -Mild diuretic.

-Long acting penicillin for prophylaxis against streptococcal lymphangitis.

-Hetrazan 50 mg tds (for filarial cases). It kills or sterilizes the adult worms.

II- Surgical treatment:

A- Bridging operations: For early cases: -.

Aim: to create a new channels to drain the lymph.

1-Omental pedicle flap:

A pedicle flap of omentum is laid in the thigh muscles aiming at development of lymphatic collaterals that drain the lymph through lymphatics of omentum.

2-Micro-lymphatico-venous shunt:

Microsurgical anastomosis between small lymphatic vessels & S.C venules.

3-Micro-lymphatic transfer (LN transfer):

Normal lymphatics are taken from the healthy limb and implanted at the site of lymphatic obstruction of the diseased limb in order to bypass the obstruction

4- Lymphangio-intra osseous anastomosis:

Removing a long wedge from the chin of the tiba in order to drain lympatics directly into the bone marrow

5-Kondoleon's operation:

Removing a long strip of the deep fascia to drain the skin & S.C lymph into deep lymphatics.

Sistrunk's operation:

B- Excision (debulking) operations: For late cases :.

Aim: excision or debulking of the hypertrophied tissues to reduce the size & weight of the limb

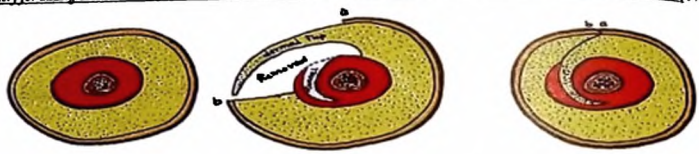
1-Sistrunk's operation: modification of Kondoleon operation.

-A large strip of skin, S.C tissue & deep fascia removed from outer aspect of the limb then closure of the skin & SC. tissue over the muscle.

-The same procedure is repeated on the inner side two months later.

2- Thompson's (Swiss roll) operation:

It resembles Sistrunk's operation but excess skin is not removed but its epidermis is shaved out and the resultant dermal flap is buried into the muscle like Swiss roll aiming to create new lymphatic shunts between the subdermal lymphatics & deep lymphatics.



Thompson's (Swiss roll) operation

3- Charle's (flaying) operation:

The skin, SC. tissue & deep fascia are removed and the bared muscles are covered by split skin grafts obtained from removed skin It has bad cosmetic results yet has no recurrence because the epidermis has no lymphatics

III- Amputation :

It is indicated in severe cases with grossly heavy limb causing disability

Lymphadenopathy

Definition:

Any disease affecting lymph nodes and causes their enlargement.

Aetiology:

I-Infection:

Bacterial, T.B, syphilis, Brucellosis, & Typhoid.

Viral: viral hepatitis, & infectious mononucleosis.

Parasitic: Toxoplasmosis.

II-Blood diseases:

Proliferative: leukaemia (lymphatic) & myelofibrosis.

III-Metabolic diseases:

Lipoid storage diseases: Gaucher's disease & Niemann pick's disease.

IV-Collagen diseases:

Felty's syndrome, Still's disease & systemic lupus erythromatosis.

V-Neoplastic:

A-Primary: Hodgkin & non-Hodgkin lymphoma.

B-Secondary: metastases (much more common).

Tuberculous lymphadenitis

There are two distinctive patho-clinical types:

1-Lymphatic born T.B. lymphadenitis

(Fibrocaseous type)

Aetiology:

I-Causative organism: Mycobacterium tuberculosis usually the bovine type.

II-Route of infection: lymphatic borne.

III-Predisposing factors: bad general condition, & poor resistance.

Pathology:

Site: The commonest site is upper deep cervical lymph nodes from TB of the tonsils.

Other sites include: -Lower deep cervical L Ns from upper deep cervical LNs.

-Mediastinal & axillary LNs from pulmonary T.B.

-Mesentric LNs (tabes mesentrica) from intestinal T.B.

Types : There are two types according to virulence of the organism & resistance of the patient.

1-Fibrous type: (common in adults)

-If the resistance of the body overcame the virulence of the organism.

-There is dense fibrosis of the L.N without caseation.

2-Caseous type: (common in children)

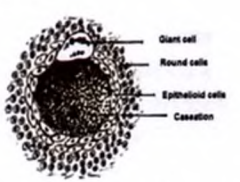
-If the virulence of the organism overcame body resistance.

-The characteristic microsopic tubercle is formed of :

1-central zone of caseation.

2-Midzone of epithelioid cells & Langehan's giant cells.

3-peripheral zone of small round cells.



Microscopic picture of the tubercle

Complications:

1-Cold abscess: due to fusion of multiple caseating foci. It may be complicated with:

a- secondary bacterial infection.

b- rupture forming T.B sinus or ulcer.

c- pressure on important structures e.g. common bile duct, mediastinal syndrome.

2-Fibrosis: it causes traction on the surrounding structures e.g. oesophageal traction diverticulum

3-Calcification.

Incidence:

The commoner caseous type affects mainly children & the fibrous type occurs mainly in adults.

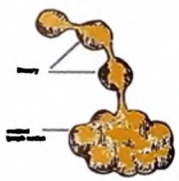
Clinical picture: -

I-General : TB toxaemia is usually absent.

II-Local:

A-Localized lymphadenopathy:

-A group of LNs usually the upper deep cervical LNs are *matted* together (due to periadenitis). They can be counted due to persistence of grooves among them .The intervening lymphatics between relatively wide apart LNs may be fibrosed and form cords between LNs. The latter feel like *rosary beads* (pathognomonic).



Fibrocaseous TB. LNs

-The consistency is firm unless caseating (soft cystic) or calcified (hard).

B-Cold abscess:

It is neither cold (It is warm) nor an abscess (It contains caseous material not pus). The name cold is attributed to absence of inflammatory signs i.e. hotness ,redness,& tenderness. It may be **collar stud** shaped i.e. with superficial and deep subfacial parts connected by narrow track

C- TB. Sinus:

Typical TB ulcer with undermined edge, cyanotic margin, soft base & pale granulation tissue dicharaging serous or seropurulent fluid if secondary infected.

Investigations:

1-Blood picture: anaemia, leucopenia with lymphocytosis (relative).

2-Tuberculin test: good negative test.

3-ESR: for confirmation & follow up.

4-Aspiration of cold abscess: by valvular manner.

The specimen is sent for
- Ziehl Nelson's stain
- Culture on Lowenstein Jensen media.
- Guinea pig inoculation

5-Lymph node biopsy: for histopathological examination.

Differential Diagnosis:

From other causes of lymphadenopathy.

Treatment:

1-Conservative treatment: All cases should receive antituberculous drugs:

1-Sanatorium admission.

2-Improve the general condition.

3-Anti-tuberculous drugs.

II-Surgery: indicated in

1-Persistent localized lymphadenopathy in spite of treatment for 6 months.

2-Persistent T.B ulcer or sinus or cold abscess.

Technique:

Excision of all lymph nodes including abscess or overlying ulcer or sinus and the wound closed without drain.

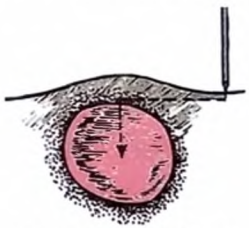
III Treatment of complications:

A-Cold abscess:

1-Aspiration: repeated aspiration through

- a healthy skin away from the abscess.
- a non-dependent site.
- in a valvular manner.

To avoid development of TB. sinus.



Aspiration of cold abscess

2- Infected cold abscess needs the usual drainage.

3-Resistant cold abscess may need excision with the underlying LN(s).

B- T.B sinus & ulcer:

-Repeated dressing with streptomycin powder.

-Resistant ulcer may need excision with the underlying LNs.

Blood Borne TB lymphadenitis

(Lymphadenoid type)

Aetiology:

The organism reaches the LNs through the blood.

Pathology:

Site: Many groups of LNs allover the body

-The organism invades the lymph nodes from the hilum so, the medulla is mainly affected there is no matting (no periadenitis) , no caseation, & no cold abscess or sinus.

-Microscopically: Scanty giant cells and *No caseation* .

Incidence:

It is more common in elderly people.

Clinical picture:

General: T.B Toxaemia.

Generalized lymphadenopathy: LNs are enlarged, firm rubbery (lymphadenoid), dis mobile, with smooth surface.

Investigations:

Investigations for generalized lymphadenopathy (see lympoma).

Treatment:

Conservative ttt as before. Surgery is indicated only for L.N. biopsy.

Lymphoma

Definition:

Neoplastic lesions of the lymphoid tissue not involving the peripheral blood.

Types:

There are two distinctive types , Hodgkin's & Non Hodgkin's lymphoma.

Hodgkin's lymphoma

Definition:

A pleomorphic malignant neoplasm which arises from lymphoid tissue.

Pathology:

Site: From any lymphoid tissue in the body:

Nodal: The commonest is the lower deep cervical LNs.

Extra-nodal: spleen, liver, bone marrow, lung & intestines.

MAC: *LNs:* are enlarged, firm rubbery, discrete, cut section is grayish pink.

Spleen: may be enlarged with grayish pink deposits & irregular surface.

MIC: *Pleomorphism* .There are **four** types of cells :

1-Reticulum cells (histocytes).

2-lymphocytes & plasma cells.

3-polymorphnuclear leukocytes & oesinophils.

4-*Dorthy Reed Sternberg's cells*. They are **4** nucleated-mirror image giant cells. They arise form reticulum cells, which undergo nuclear division without cytoplasmic division.

- According to the predominance of different cells in the tumour, there are **four** histopathological types (classification):

1-Lymphocyte predominant type: (the best prognosis).

2-Nodular sclerosis type: (the commonest), there are broad fibrous bands that disturb the LN architecture (good prognosis).

3-Mixed cellularity type: there is a festival of the all above cells.

4-Lymphocytes depletion type: (the worst prognosis).

Staging: (*Ann Arbor staging)*

It is a clinical staging. There are four stages:

Stage I: Single involved group of lymph Node (I) or single extralymphatic site (I_E).

Stage II: More than one group of lymph nodes are involved on one side of the diaphragm (II).

If an extralymphatic site on the same side of the diaphragm is involved → stage (II_E).

Stage III: Involvement of LNs on both sides of the diaphragm:

-If spleen is involved it is stage (III_S).

-If one extralymphatic site is involved it is stage (III_E).

Stage IV: Extensive extralymphatic spread i.e liver, intestine, lung, and bone.

*All stages are subdivided into either A or B according to absence or presence of systemic symptoms (fever, night sweats & loss of weight more than 10% in six months) respectively.

N.B. Extralymphatic site is any lymphatic tissue site other than lymph nodes, spleen & Waldeyer's ring (lingual tonsils, palatine tonsils & adenoid).

Complications: (four).

Complications are due to infiltration of different organs in various body regions.

1- Abdominal :

- Splenomegaly (75%) with possible hypersplenism.
- Hepatomegaly (50%) may be with jaundice.
- Ascites: due to peritoneal infiltration or hepatic dysfunction.

2- Chest:

- Mediastinal syndrome: dyspnea, cough & pain (enlarged mediastinal LNs) .
- Pleural effusion.

3- Skeletal:

- Bony aches
- Pathological fracture e.g. collapse of the vertebrae .

4- Neurological:

-Peripheral neuropathy - Root compression in vertebral collapse.

Incidence:

Hodgkin's lymphoma has two peaks of age incidence (15-40 years) and (over 50 years).

Males are more affected than females.

Clinical picture: (four)

1- General: are due to systemic manifestations:

1- Fever: *Pel-Ebstein fever* is an irregular fever (2weeks of fever & 2 weeks of freedom.

2- Night sweats, weight loss & pruritis.

II- Lymphadenopathy:

- Single or multiple lymph node groups are involved .
- *Site:* lower deep cervical LNs.
- *Size*: variable size with the largest one in the center.
- *Shape:* rounded or oval .
- *Surface*: smooth.
- *Skin over*: free .
- *Surrounding structures*: usually not fixed
- Consistency: firm elastic (rubbery).

III- Abdominal examination:

- Hepatomegaly, splenomegaly & ascites.

IV- The patient may present with complications.

Investigations: (for generalized lymphadenopathy).

I- Laboratory investigations:

1- Bl. picture: It is essential to
- rule out leukaemia (↑↑leucocytic count.)
- suggest T.B. (relative lymphocytosis)
- suggest Hodgkin (anaemia & oesinophilia)

It may show pancytopenia, either due to hypersplenism or bone marrow infiltration.

2- Bone marrow picture (Sternal puncture): To diagnose aleukaemic leukaemia and can detect the cause of pancytopenia

3- Liver function test: S. alkaline phosphatase & 5'nucleotidase.

4-Serological tests to rule out infective diseases(TB.,typhoid, syphilis & brucelosis):

Tuberculin test, Widal test, VDRL.....,etc

II- Imaging studies:

1- Plain X ray chest may show:

- pulmonary TB. & calcified hilar LNs
- enlarged mediastinal LNs.
- lung infiltration or pleural effusion.

2- Ultrasound, C T scan , & MRI :

They may detect hidden lymph nodes, organomegaly & ascites.

3- Positron emission tomography (PET) and. The system detects pairs of gamma rays emitted indirectly by a positron-emitting radionuclide (tracer), which is introduced into the body on a biologically active molecule. It depends on the unique metabolic activity of the tumor to concentrate the tracer. In modern PET-CT scanners, three-dimensional imaging is often accomplished with the aid of a CT X-ray scan performed on the patient during the same session, in the same machine.

4- Isotope scan :

Liver, lung & bone scan.

III- Invasive investigations:

*1- Lymph node biopsy:fine or trucut needle or better surgical biopsy is m*andatory to prove the diagnosis.

2-Staging laparotomy (rarely done nowadays): Exploration of the abdomen and :

* Splenectomy (ttt of hypersplenism & avoid radiation injury to Lt. kidney & lung).
* Liver biopsy (both lobes).
* LNs. biopsy (mesentric, retroperitoneal).
* In ♀ the ovaries are fixed in Douglas pouch in the midline to protect them from irradiation.
* Bone marrow biopsy from iliac crest.

-The rapid progress of non-invasive investigations e.g. MRI makes the value & need for staging laparotomy to regress. However, laparoscopy nowadays is very beneficial.

Differential diagnosis:

From other causes of generalized lymphadenopathy especially lymphadenoid TB.

Treatment:

Stage I & stageIIA: Radiotherapy alone.

A- Supradiaphragmatic: (*Mantle irradiation*) .

(4000 rads over 4 weeks) to cervical, axillary & mediastinal LNs.

B- Infradiaphragmetic: (Inverted Y field irradiation) .

To the para-aortic, iliac, pelvic & inguinal LNs ± splenic axis irradiation (If not removed) .

Important organs are protected with lead shields.

Stage IIB, III & IV: combination chemotherapy & radiotherapy.

* Combination chemotherapy (Quadriple MOPP therapy).Mustine (Nitrogen Mustard), Oncovin (vincristine), Procarbazine & Predinsolone (corticosteroid) or COPP therapy replacing Mustin by Cyclophosphamide .

-Predinsolone may bring rapid relief of systemic manifestations (Pruritis & anaemia).

-Chemotherapy is given in cycles of 6 weeks (2weeks of therapy & 4 weeks of rest) to release bone marrow cells & GIT epithelium from suppression of chemotherapy.

Prognosis:

The prognosis is good. It depends on the histological type and the stage of the disease.

The overall 5 years survival rate is about 80%.

Non Hodgkin's lymphomas

Definition:

They are groups of malignant neoplasms that arise from a monoclonal proliferation of a malignant cell of lymphoid origin.

Pathology:

Site: From any lymphoid tissue in the body:

Nodal: the commonest is the upper deep cervical lymph nodes.

Extranodal: (more common than Hodgkin) as liver, spleen, bone marrow, & bowels.

MAC: -The lymph nodes are amalgamated (fusion with loss of boundaries).

-The consistency is variable (soft, firm or hard).

-In cut section, the lymph node loses its architecture, which is replaced with white sarcomatous tissue that invades the capsule & surroundings.

-Spleen & liver are infiltrated with similar deposits.

MIC: There are four histological types according to cell of origin .

1- B cell lymphoma: which is further classified into:

a) Small cell lymphoma .

b) Large cell lymphoma.

c) Mixed small & large cell lymphoma.

d) Immunoplastic lymphoma.

2- T cell lymphoma.

3- Lymphoblastic lymphoma.

4- Histiocytic lymphoma.(reticulum cell lymphoma)

Complications:

As in Hodgkin but extralymphatic complications are commoner e.g. gastric lymphoma may cause gastric outlet obstruction, and intestinal lymphomas may cause bleeding or obstruction.

Incidence:

It is more common in elderly but may affect adults.

Clinical picture:

I- General: Malaise, weight loss, organomegaly and abdominal mass.

II- Lymphadenopathy:

-Single or multiple LN groups (of variable size) .

-*Site:* commonly the upper deep cervical LNs. -Size: variable .

-*Shape & surface*: LNs are amalgamated together (They can't be counted) form a large mass with irregular or bosselated surface) .

-*Skin over*: is stretched with dilated or apparent veins and may show fixation or ulceration.

-*Surrounding structures*: may be infiltrated. -*Consistency*: variable.

III- The patient may present with complications.

Investigations :

As before.

Treatment:

*Combination chemotherapy CVP Cyclophosphamide + Vincrestin + Predinsolone and Bleomycin

*Radiotherapy.

It is more radiosensitive but more radiorecurrent .

*Surgery is indicated only in:

a) LN biopsy.

b) Excision of an early localized disease (doubted) .

c) Dealing with surgical complication e.g. intestinal bleeding , & obstruction .

Prognosis:

Very bad.

Burkitt's lymphoma

Aetiology:

- It arises from B-lymphocytes
- It is caused by *Ebstein Barr (EB) virus* which causes infectious mononucleosis when it infects healthy individual. When infection is for a ch. malarial patient, it may induce this form of lymphoma.

Pathology:

It commonly affects the jaw, kidney, ovaries, LNs, spleen.

Incidence:

It usually affects children below 15 years. Common in eastern Africa.

Clinical picture:

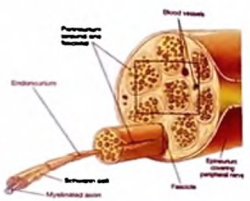
It presents with progressive painless swelling of the jaw that distorts the face displacing the eye & protrudes in the oral cavity.Other organ affection may be manifested.

Treatment:

By combination chemotherapy & radiotherapy (good response).

The nerves

Basic anatomy of nerve



Neuron consists of cell body, associated dendrites and usually one axon. Nerve trunk is composed of large number of nerve axons, which are gathered and supported by connective tissue. All axons are surrounded by Schawn cells. In myelinated fibers, Schwan cells rotate several times around the nerve axon to form an insulating sheath called *the myelin sheath*,. The myelin sheath is interrupted between Schwan cells at what is called *nodes of Ranvier* The basement membrane of Schwan cells with endoneurial collagen fibers form an *endoneurial sheath*. A large number of nerve fibers are gathered in fascicles surrounded by a connective tissue sheath called *perineunium*. The whole fascicles are bounded together and surrounded by another connective tissue sheath called *epineurium* which represents the outer capsule of the nerve trunk

NERVE INJURY

Aetiology:

I- Open trauma:

Stab, bullet or deep burn

II- Closed trauma

1-Fracture of bones and dislocation of joints

2-Compression of the nerve against bone by tourniquet or tight plaster

3-Traction injury during reduction of bone fracture & birth

4-Chemical injury: through I.M injection of irritant drugs leading to intense fibrosis

5-Ischaemic injury: due to injury of the main vessels

Pathology:

Seddon's classifications:

Injury to the nerve may lead to

1-Neurapraxia: (Nerve concussion) :

- Temporary loss of function due to localized demyelination of a segment of the nerve but the nerve axons are not interrupted.
- It is caused by mild compression e.g. tourniquet or stretch e.g. dislocation.
- Nerve conduction distal to the injury remains normal.
- Spontaneous complete recovery occurs in hours, days or weeks provided that the cause is removed

2- Axonotmesis:

- Rupture of nerve axons within intact endoneurial sheaths.

- *Wallerian degeneration* occurs in the distal parts of ruptured axons. The axons break into fragments & myelin sheath is disintegrated into fat droplets, which are engulfed by phagocytes. Where the Schwan cells increase in size, undergo mitosis & persist as empty neurolemmal sheaths
- Retrograde degeneration occurs in the proximal parts of the neurons. The cell swells & becomes oval & loses its dendrites. The nucleus becomes eccentric. Similar changes to Wallerian degeneration occur in the proximal axons only to the nearest node of Ranvier
- Regeneration occurs slowly by down growth of the proximal axon at a rate of about 1-2 mm per day till the axons reach their nerve endings
- As the neurolemmal sheathes are intact, so, regeneration occurs perfectly with full recovery.

3-Neurotemesis:

- Partial or complete division of a nerve trunk leading to interruption of both nerve axons & neurolemmal sheath
- Wallerian & retrograde degeneration occur in the severed neurons
- As the neurolemmal sheaths are not in alignment with their proximal axons, so regeneration is usually incomplete, imperfect & may be associated with neuroma formation (see below)

Complications:

1-Motor complications:

1-Paralysis & inability to perform certain movement.

2-Muscle disuse atrophy may end in fibrosis.

3-Deformity due to unopposed action of the unparalyzed muscles.

2-Sensory complications:

1-Anaesthesia, lack of sensation

2-Trophic ulcer e.g. cigarettes ulcer

3-Trophic changes:

-They may be due to sensory loss, disuse atrophy or vascular changes.

They include

- *Thin smooth skin.
- *brittle fissured nails
- *tapering of the fingers
- *trophic ulcers

Terminal neuroma

Lateral neuroma

Fusiform neuroma

4-Complications of regeneration

1-Neuroma formation:

It is a localized very tender swelling at the site of nerve injury caused by regenerating mal-directed nerve axons. It may be

a)Terminal neuroma: after complete division

b)Lateral neuroma: after partial division

c)Fusiform neuroma: may follow nerve contusion with axonotemesis ,but fibrosis prevents the descent of regenerating axons

2-Maldistribution & faulty regeneration

-They commonly occur in mixed nerves

-The nerve fibers don't regenerate in their proper sheaths leading to poor function

-Also, poor results occur in nerves supplying many small muscles performing fine movement

Clinical picture:

-History of trauma

1-Motor manifestations

1-Loss of voluntary contractions.

2-Apparent deformity.

3-Loss of reflexes.

2-Sensory manifestations:

1-Loss of sensation in an area less than that anatomically supplied by the nerve due to anatomical overlap of nerves

2-Pain or paraesthesia in the area of nerve distribution

N.B. Causalgia is intense burning pain that results from incomplete major nerve injury It is thought to be due to formation of false synapses between sympathetic and sensory nerve fibres

3-Vasomotor manifestations:

-Early, the denervated area is red & warm (paralysis of sympathetic fibers)

-Later, it becomes blue & cold due to vasoconstriction, which may be caused by hypersensitivity to circulating catecholamines (denervation hypersensitivity)

4-Sudomotor manifestations: *"from Latin sudor = 'sweat'*

-Loss of sweating (anhydrosis) of the denervated area of the skin

5-The patient may present by complications

Special tests

**Sweating test:* dusting the body with quinizarin powder and give the patient aspirin tab & a cup of coffee to induce sweating. Sweat causes the dry powder to turn to deep purple colour. The anhidrotic area remains unchanged

2-Tinel's test: to detect the level of regeneration, light percussion along the course of the nerve is applied from distal to proximal till a tingling sensation is felt by the patient

Investigations:

1-Nerve conduction velocity (NCV):

-Normally NCV is 50-70 m /sec

-It is decreased in nerve compression & injury but, it is normal in neuropraxia.

2-Electromyograhy (EMG):

-Normally muscle contraction causes electric activity above 500 mVolt.

-Few weeks after nerve injury, there are weak rhythmic twitches of the denervated muscle (1-10 times/ sec and less than 200 m Volt.)

3- Magnetic resonance imaging (MRI):

-It is a promising imaging to display peripheral nerve injury and expected to be a routine investigation in the future. Its value is maximum in cervical nerve roots imaging after brachial plexuses injury.

Treatment:

I-Conservative treatment:

Indicated in closed injuries when neuropraxia or axonotemesis are suspected

- The aim is to prevent muscle wasting and stretching, deformity and stiffness of joint. It consists of

(1) *Splinting*: to prevent stretching of the paralyzed muscles but permit free movement of the unparalyzed muscles. The splint should be removed at least once a day to permit all joints to move through the whole range of passive movement

(2) *Electro-therapy*: Galvanic current stimulation of the paralyzed muscles is essential to minimize wasting . At least 50 strong contractions of each paralyzed muscle should be elicited every day starting as early as possible after nerve injury

(3) *Physiotherapy*: Active and passive exercise together with gentle massage are performed regularly to maintain joint movement, minimize fibrosis, maintain tissue nutrition & prevent oedema

(4) *Protection of skin* against injury and heat in the anaesthetic areas to avoid trophic ulcers

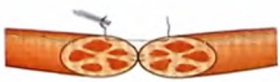
II- Operative treatment:

It is indicated in

(1) Open injury.

(2) Closed injury with no signs of regeneration under conservative measures within 2 months

(3) palpable neuroma



Simple epineurial repair

Primary repair:

- In clean tidy wounds, immediate primary nerve repair is done by simple suturing of the ends.
- The essence of good nerve repair is accurate coaptation of the ends without tension in a healthy bed

Secondary repair:

- In late cases, the nerve is explored. The ends are trimmed with a sharp scalpel till healthy fascicles appear. then do nerve suturing if there is no gap or nerve grafting if there is a gap

A- Nerve suturing is done with 6/0 prolene suture *by epineural repair*: the sutures spick up the nerve sheath epineurium only

B- Nerve grafting. Long segment of a cutaneous unimportant nerve such as *the sural nerve*.is harvested and then divided into fragments, whose length matches

Cable graft

with the length of the defect. Each fragment is sutured to a well matched fascicle in both ends. this type of multiple nerve grafting is called *(cable grafi)*

III-Secondary procedures:

Indications:

1- Neglected and longstanding cases

2- Failed or poor results after nerve repair.

3- Cases with fibrosed muscle or skeletal

Aim:

To improve function and correct deformity

Types:

1- Muscle and tendon transfer: either from nearby healthy muscles or use of free muscle flap from distant area by microvascular technique.

2- Arthrodesis: Fixation of joints in functioning positions

3- Osteatomy to correct skeletal deformities

Prognosis

The prognosis of nerve injury and repair depends on many factors

1- Severity of injury: according to Seddon classifications, neurotemesis is the worst. Also, clean cut injury is better than crushed injury

2- Level of injury: Proximal lesions do worse than distal ones. However suturing proximal main trunk appears easier and more satisfactory.

3- Type of nerve: classically, mixed nerves have worse prognosis than pure sensory or pure motor ones, Also, motor nerves that supply large muscles perfrming coarse movement such as radial & femoral nerves have better prognosis than those supplying small muscles with fine precise movement (hand)

4- Associated injuries: No doubt that associated bone, soft tissue and vascular injures add more burden on the prognosis. Repairing these structures may improve the outcome

5- Delay in repair: early repair of nerve injury when possible gives the best results. The longer the period of muscle paralysis the more the wasting and the less the chance of complete recovery.

6- The age of patient & the general condition: childen have better prognosis than adults & old patients.

I- Upper limb nerve injury

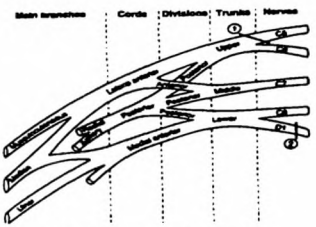
Brachial plexus lesions

Types:

A- Erb Duchenne's paralysis (upper trunk lesion).

Definition:

It is injury of the C5 and C6 roots.



The brachial plexus injury

Aetiology:

I- Traction injury:

1- Forcible widening of the angle between the head and the shoulder this typically occurs in faulty traction on the baby's arm during delivery (birth injury).

2- Weight falling on the shoulder causing its pressure injury .

II- Open injuries: Stab injury or gunshot injury.

Birth injury

Clinical picture:

1- Motor loss: Paralysis of abductors and external rotators of the shoulder, flexors of the elbow and forearm supinators. This paralysis leads to *policeman tip deformity* where the upper limb is held adducted, internally rotated, the elbow is extended & the forearm is pronated with the wrist and fingers flexed.

2- Sensory loss over the lat. side of the arm & forearm.

Traction injury

B- Klumpké parlyis (lower trunk lesion):

Definition:

It is C8 and T1 roots injury.

Aetiology:

1- Cervical rib and scalene syndrome. 2- Pancoast tumour.

3- Hyperabduction of the upper limb as dragging along the ground by the arm damage.

Clinical picture:

1- Motor loss: - Paralysis of flexors of the writs and fingers. This results in claw hand deformity that resembles combined median and ulnar nerve deformity.

2- Sensory loss: Anesthesia along the inner side of the forearm and the inner 3½ fingers.

3- Autonomic manifestations *(Horner's syndrome)* : Injury to the preganglionic fibers in T1 root leads to ipsilateral ptosis, miosis, anhydrosis of the face & enophthalmos.

C) Whole plexus injury

It is a rare type because an injury sufficiently severe to damage all the roots of the plexus is expected to cause serious injuries on adjacent vital structures and usually lead to rapid death. However some cases are reported

Clinical picture:

The whole limb is paralyzed and flaccid with complete anaesthesia except the inner side of the upper arm supplied by the intercostobrachial nerve (T2) and the upper part of the lateral aspect of the shoulder supplied by the supraclavicular nerves (C4). Horner's syndrome may also be present

Investigations:

1. NCV& EMG to study the degree of muscle affection
2. Chest and neck X rays for detection of Pancoast tumour, cervical spine fracture or disease
3. CT Myelography to detect root avulsion
4. MRI is very helpful to confirm lesions of the roots.

Treatment:

I-Conservative treatment:

It is indicated in closed injuries for the hope of spontaneous recovery. It follows the some line described with special considerations.

1-The arm is splinted in right angle abduction and the forearm is flexed and supinated.

2-Stiffness is prevented by passive & active exercise.

3-Electro-therapy is essential to minimize wasting of muscles.

II- Surgical treatment:

1- Nerve repair:

It is indicated in

a. Open injuries provided that the general condition of the patient permits.
b. Closed injuries which failed to show any evidence of recovery within 2 months especially when a neuroma develops and becomes palpable.

The nerves are explored, identified, trimmed and sutured as described before

2-Secondary procedures:

a) Latissmus dorsi transfer to imrove shoulder abduction & external rotation

b) Osteotomy of radius to improve the appearance of pronation deformity of the forearm.

Prognosis:

The results up till now are not satisfactory but the prognosis in children is better

1- The Circumflex nerve injury

Aetiology:

1- Fracture surgical neck of the humerus.
2-Shoulder dislocation
3- Crutch palsy
4- Stab and gunshot wounds
5- Direct blow to the shoulder or upper humorus.

Clinical picture

I- Motor loss

The paralysis of the deltoid muscle causes inability to abduct the arm and *flat shoulder deformity* with prominent acromion

II- Sensory

Area of anaesthesia over the posterior axillary fold

flat shoulder deformity

Treatment:

Usually conservative treatment as before

2- The long thoracic nerve
(Nerve of Bell)

Aetiology:

1- Operation on the axilla e.g. radical mastectomy or chest wall
2- Stab or gun shot

Clinical picture:

Paralysis of the serratus anterior muscle causes inability to raise the arm above the head as in hair dressing and *winging of scapula* in forceful protraction movement

Winging of the scapula

Treatment:

Usually conservative as before.

3- Radial nerve Injury

Aetiology:

I- Open trauma: e g cut wounds & gunshots.

II- Closed trauma:

1-Fracture of humeral shaft. Also ,fracture radial head may injure the post. interosseous nerve

2- Compression of the nerve by:

a) crutches. b) tourniquet. c) edge of the operating table.

d) edge of kitchen chair during sleep durnken in sitting position *(Saturday night paralysis)*

3- Injection of irritant drug in the back of the arm .

Clinical Picture:

I- Injury below the elbow:

The nerve is purely sensory after giving the posterior interosseous nerve.

A- No motor loss.

B- Sensory loss: It is usually negligible in the from of anaesthesia over the anatomical snuff box. Injuries in the lower forearm may cause more sensory loss than injuries in the upper forearm because the radial nerve joins the lateral cutaneous nerve of the forearm at the lower forearm so ,lower injuries might damage both.

N.B: injury to posterior interosseous nerve causes:

- Inability to extend the fingers *" fingers drop"* while active extension of the wrist is possible by extensor carpi radials longus (supplied by the radial nerve above the elbow).
- No sensory loss as the nerve is purely motor.

II- Injury at the spiral groove: Injury to posterior interosseous nerve +.

A- Motor loss: Inability to extend the wrist *" wrist drop"* but active extension of the elbow is still possible by the long head of triceps (supplied by the radial nerve in axilla).

Wrist drop

B- Sensory loss: as before.

III- Injury at the axilla:

A- Motor loss: *as before* +.

- Inability to extend the elbow actively due to paralysis of triceps & anconeus muscles.

Area of anaesthesia

B- Sensory loss: *as before* +.

- anaesthesia in the lower lateral aspect of the arm and back of the forearm.

Treatment :

I- Conservative ttt:

-It is indicated in closed injury (neuropraxia & axonotemesis). It consists of:

1-Galvanic stimulation to minimize wasting & avoid fibrosis of muscles.

2-Active & passive exercise to avoid stiffness of joint.

3-Splint to prevent shortening and permanent deformity.

II- Surgical ttt is indicated in:

- open injuries.
- failed conservative ttt i.e. no sign of recovery within 6 weeks.

•It includes:

1-Nerve suturing or nerve graft.

2-Tendon transfer (if regeneration failed). Transfer of some flexor tendons to tendons of the extensors of the fingers, thumb & wrist.

Tendon transfer

Prognosis:

The prognosis is good because:

- It is almost pure motor nerve.
- It supplies coarse muscles with unskilled movement.
- Even if regeneration fails tendon transfer is easy & gives good result.

4- Median nerve Injury

Aetiology: -

1- Open trauma: e.g. cut wrist, bullet injury, and stab wounds.

2-Closed trauma:

1- Fracture, dislocation: Colle's fracture, supracondylar fracture & dislocation of the elbow.

2- Compression on the nerve: e.g. carpal tunnel syndrome.

Clinical picture:

Ape hand deformity

I- Injury at the wrist:

A- Motor loss:

1-Paralysis of the thenar muscles except the adductor pollicis (supplied by the ulnar nerve) this results in wasting of the thenar eminence.

2-*Ape hand* or *simian(قردي) hand deformity:* The thumb is extended & adducted by the unopposed adductor pollicis so, it lies in the same plane of the other finger like Ape hand.

B- Sensory loss:

There is anaesthesia over the lateral 2/3 of the palm and the lateral 3½ fingers on their whole palmer surfaces and dorsal surface of their distal phalanges with possible cigarettes burn.

II- Injury around the elbow

A- Motor loss: as above +.

1- Paralysis of pronator teres & quadratus leading to loss of pronation.

2- Paralysis of flexor carpi radialis leading to ulnar deviation of the wrist during flexion (intact flexor carpi ulnaris supplied by the ulnar nerve).

3-Paralysis of flexor digitorum superficialis & the lateral ½ of the flexor digitorum profundus leading to failure of flexion of the index and ring fingers = *Pointing index* or *benediction attitude* (منح البزكة) and can be detected by *clasping test* (failure of flexion of the index finger when doing a fist or clasping).

Benediction attitude

4- Paralysis of flexor pollicis longus & brevis leading to failure of flexion of the thumb.

B- Sensory loss : As before.

Ochsner's clasping test

***Treatment*:**

I- Conservative ttt .

Indications & lines as in radial nerve injury.

III- Surgical ttt:

Indications as before

I- Nerve repair.

II- Tendon transfers: *Bunnell's tendon transfer* .

In injury of the median nerve at the wrist .If no recovery, the opposition of the thumb can be restored by transferring the flexor superficialis tendon of the ring finger to the base of the proximal phalanx of the thumb after rerouting it around the tendon of flexor carpi ulnaris.

Bunnell's transfer

Prognosis:

Prognosis is bad because:

1- It is a mixed nerve.

2- Sensory loss causes marked disability & trophic ulcers.

5- Ulnar nerve Injury

Aetiology:

I- Open trauma: stab or bullet along the course of the nerve.

II- Closed trauma:

1- Fractures or dislocations: around the elbow.

2- friction of the ulnar nerve against the medial epicondyle in cubitus valgus deformity →delayed ulnar neuritis.

Cardboard test

Clinical picture:

Froment's test

I- Injury at the wrist

A- Motor loss:

1-Paralysis of interossei muscles →failure of adduction & abduction of fingers detected by *cardboard test*

2- Paralysis of adductor polices leading to failure of active adduction of the thumb detected by *Froment 's test* i.e. if the patient is asked to pinch a paper firmly between the thumb & fingers ,he flexes the distal phalanx of the thumb as the paralyzed adductor pollicis is compensated by intact flexor polices longus

Partial claw hand

3- Paralysis of the two medial lumbrical muscles causing partial *claw hand deformity* in the medial two fingers (the lateral two lumbricals are supplied by the median nerve).

4- Paralysis & wasting of hypothenar muscles.

B- Sensory loss:

Loss of sensations on the medial 1/3 of the palm and palmer aspect of the medial 1½ fingers

N.B. The dorsal cutaneous branch of the ulnar nerve arises about 2 inches above the wrist & usually escapes injuries at the wrist.

II Injury around the elbow

A- Motor loss: as before +

Paralysis of flexor carpi ulnaris and the medial ½ of the flexor digitorum profundus. The later makes the clawing less marked if the nerve is injured at the wrist (ulnar paradox)

B. Sensory loss

over the medial 1/3 of the palm & medial 1 ½ fingers on both palmer & dorsal surfaces

Treatment

I- Conservative ttt

The same indications & lines as before

II Surgery: indicated in

- Open injury
- Failure of conservative ttt

It consists of

1-Nerve repair in neurotmesis

Area of anaesthesia in ulnar nerve injury

2- transposition of the nerve anterior to medial epicondyle in delayed ulnar neuritis

3- Tendon transfer: if no recovery had occurred

The tendon of flexor digitorum superficialis to the ring finger is divided into strips and sutured to the extensor expansion of the claw fingers to correct the claw deformity

Prognosis

It is not bad. Only those who perform fine movement e.g. surgeons, typist & pianist suffer more

II- Lower limb nerve injury

1- Sciatic nerve injury

Aetiology:

1-Dislocation of the hip and fracture of pelvis & femur.

2-Deep wounds in the buttock and the thigh.

3-Intra muscular injection of drugs.

drop foot deformity

Clinical picture:

I- Injury in the back of the thigh .

A- Motor loss :

Paralysis of all muscles of the leg and foot. Gravity causes *drop foot deformity*

B- Sensory loss:

Anaesthesia of the leg and foot except an area along their medical aspect (supplied by the saphenous nerve from the femoral nerve).

II- Injury in the buttock:

As before plus paralysis of the hamstrings muscles that leads to weakness of flexion of the knee which can be still flexed by the sartorius and gracilis muscles.

Treatment:

I- Conservative treatment: as before, Drop foot is prevented by wearing a suitable support.

II- Surgical treatment:

A- Early cases:

Nerve repair is indicated in open injury and failed conservative treatment..

foot support

B- Late cases :

1- Persistent paralysis: great improvement of the gait is achieved by hinged-knee splint and arthrodesis of the ankle.

2- Persistent severe pain or resistant trophic ulcerations: amputation may be indicated to get rid of useless, harmful limb.

2- Lateral popliteal (Common peroneal) nerve injury

Aetiology:

I- Open injury: Deep wounds of the thigh

II- Closed injury: 1) Fracture of the upper end of the fibula.

2).Iatrogenic injury: pressure by tight strapping , Thomas splint and plasters.

Clinical picture:

I- Motor loss:

Paralysis of the extensor and peroneal muscles. Early foot *drop* develops later *talipes equinovarus deformity* ensues due to action of the unopposed plantar flexors and invertors

II- Sensory loss:

Anaesthesia in the dorsum of the foot and the 2/3 of the leg except along their medial aspect

Treatment:

I- Conservative treatment:

The same indications & lines as before. Drop foot is prevented by wearing a suitable support.

II- Surgical treatment:

It is indicated in open injuries and failure of conservative ttt.

Prognosis:

The prognosis is better in injuries in the thigh & popliteal fossa than in lower injuries.

3- Medial popliteal (Tibial) nerve injury

Aetiology:

It is rarely injured due to its deep position supported with muscles however, it may be injured in

I- Open injury: deep wounds in the popliteal fossa or the calf.

II- Closed injury: fracture of lower femur or upper tibia.

Clinical picture:

I- Motor loss:

Paralysis of the calf muscles (the plantar flexors of the foot and toes) and muscles of the sole . This leads to *paralytic calcaneo-valgus deformity* with clawing of the toes due to the unopposed action of the dorsiflexors and evertors of the foot .

II- Sensory loss:

- Anaesthesia with severe trophic ulcers of the sole of the foot. Causalgia is also common.

Talipes calcaneovalgus

Treatment:

I- Conservative treatment:

As before:

II- Surgical treatment:

It is usually indicated as return of sole sensation is a great aim on the the expense of any sacrifice

Entrapment neuropathy

Carpal tunnel syndrome

Definition :

Compression of the median nerve and the flexor tendons under the flexor retinaculum in the carpal tunnel in front of the carpal bones. The ulnar nerve and the palmar branch of the median nerve escape compression as they pass superficial to the flexor retinaculum.

Aetiology:

Predisposing factors

1- Pregnancy & myxoedema due to increase tissue fluid deep to the flexor retinaculum.

2- Rheumatoid arthritis affecting the synovial sheaths of flexor tendons.

Incidence:

- It is more common in middle age female.
- It is bilateral in more than 50% of cases

Clinical picture:

Symptoms:

- Pain and tingling over the distribution of the median nerve in the hand. It becomes worse
  - * at night
  - * on hanging over the edge of the bed.
  - *on flexion of the wrist .

Tinel's test

Signs:

1) *Tinel's sign*: tingling and numbness on percussion over the carpal tunnel

2) *Phalen test*: Pain is elicited or exaggerated by full flexion of the hand for one minute.

3) Wasting of the thenar muscles and anaesthesia or altered sensibility over lateral 3½ fingers.

Phalen test

Investigations:

- Nerve condition velocity (NCV): It is delayed.

Treatment:

I- Conservative treatment:

Injection of corticosteroids with or without local anaesthetics may relieve pain.

II- Surgical treatment:

Division of the flexor retinaculum to release the median nerve is ideal and most effective

Neoplasms of nerves

A- Benign Tumours

1- Neurofibromatiosis

A- Solitary neurofibroma:

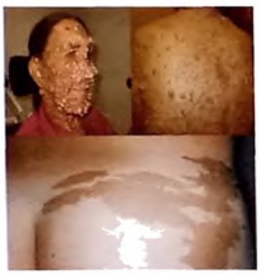
- It forms firm tender oval or rounded swelling with smooth surface
- It arises along the course of nerve trunk and moves across but not along the nerve
- It may turn malignant predisposed to by

  1- repeated irritation & trauma 2- incomplete resection

B- Generalized neurofibromatosis:

(Von Ricklinghausen's disease of nerve)

- It is a familial disease (autosomal dominant trait)
- Multiple neurofibromas of the peripheral nerves with café au lait pigmentations of the skin
- It has the same clinical picture & complications as solitary form



Von Ricklinghausen's disease

C- Plexiform neurofibroma: ***(Pachydermatocele)***

- There is diffuse fibrous thickening of the nerve branches in the skin " pachy = thick
- It commonly affects branches of the trigeminal nerve
- It causes diffuse thickening and pigmentation of skin, which becomes redundant and pendulous .

Pachydermatocele

D- Elephantiasis neuromatosa:

Elephantiasis neuromatosa

-There is diffuse thickening & fibrosis of the skin & S.C tissues simulating those of an elephant

- It commonly affects the extremities especially the lower ones
- It appears in childhood

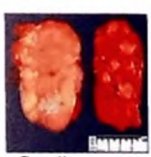
2- Schwanoma

- It is a benign tumour arises from Schwann cells and has no malignant tendency
- It forms single firm mass along the course of nerve and stretches the nerve over it
- The commonest site is 8th cranial nerve as it passes the internal auditory meatus at the cerebellopontine angle *(acoustic neuroma)* It causes functional disorders of the 5th, 6th, 7th &.8th cranial nerves and may lastly compress on the cerebellum & brain stem

Schvanoma Vs neurofibroma

3- Ganglioneuroma:

- It is a benign tumour arising from the sympathetic trunks or suprarenals
- It occurs in children and may reach huge size (spindle shaped infant)
- It forms well capsulated rounded or lobulated mass in the retroperitoneum
- Surgical removal by enucleation is easy & curative



Ganglioneuroma

B- Malignant Tumours

1- Neurofibrosarcoma:

- It may arise do novo or on top of neurofibroma
- Malignant changes are suspected when there is rapid increase in size, the tumour becomes painful & more tender, motor & sensory manifestations develope & fixity of the tumour occurs due to infiltration of the surrounding structures
- It is treated by wide safety margin excision

2- Neuroblastoma

- It is the malignant variant of ganglioneuroma and arises from the same sites
- It is a highly malignant tumour of infancy. It grows rapidly and may reach huge size *(spindle shaped infant)*
- It disseminates widely by both blood & lymphatics. Rt. adrenal tumor usually metastasizes to the liver *(Pepper's syndrome)* while the Lt. side tumour tends to metastasize to the orbit *(Hutchinson's syndrome)*
- It is treated by chemotherapy followed by wide surgical excision.

Bones
[Orthopaedic Surgery]

Traumatic conditions

Trauma may cause fracture, dislocation or subluxation.

A-Fractures

Definition:

Loss of continuity of bone by mechanical trauma (wound of bone).

Aetiology: - Trauma

I-Classic traumatic fracture:

It is caused by trauma strong enough to fracture normal bone.

- Direct trauma: over the bone.
- Indirect trauma: transmitted through the neighboring bones.

II-Pathological fracture:

Due to minor trauma to a diseased bone:-

- General disease: hyperparathyroidism, osteoporosis & rickets.
- Local disease: osteomyelitis, tumours & cysts.

III- Stress Fracture:

Due to repeated minor trauma on a normal bone e.g. march fracture.

Pathology:

Types:

I-According to the shape

Transverse **Oblique** **Spiral** **Comminuted** **Double level** **Green stick**

II-According to the integrity of the overlying skin:

1- Simple fracture with intact skin.

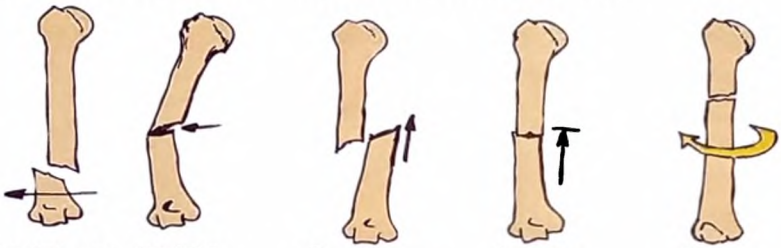
2-Compound fracture with skin wound communicating with fracture haematoma.

a) Compound from without: when trauma causes both the wound & the fracture.

b) Compound from within: when the fractured bone end injures the skin.

Displacement:

It means the deformity of the distal fragment in relation to the proximal one



Lat. displacement **Angulation** **Over riding** **Impaction** **Rotation**

-Factors affecting bone healing:

1-*Age of the patient* : young > old

2-*Vascularity*: ↓↓ vascularity →↓ healing

3-*Type of fracture* : oblique > transverse

4-*Gap of fracture*: impaction →↑ healing

5-*Immobilization* : Fixation →↑ healing

6-*Infection* : ↓↓ healing

Complications :

General complications:

1-Shock: haemorrhagic or neurogenic.

2-Haemorrhage: may be internal or external.

3-Embolism: fat embolism especially in long bone fracture.

4-Ileus: paralytic ileus may be due to reflex sympathetic stimulation.

5-Complications of prolonged recumbency:-

1-Bed sores.	2-Hypostatic pneumonia.
3-D.V.T.	4-Pulmonary embolism.
5-Osteoporosis.	6-Renal calculi.
7-Retention of urine.	8-Faecal impaction.

II Local complications:

1) Skin:	tear	infection	gangrene
2) Muscle:	tear	myositis ossificans	Volkmann's ischaemic contraction
3) Vessels:	spasm	tear	aneurysm
4) Nerves:	neuropraxia	axonotemesis	neurotemesis
5) Joint:	dislocation	haemoarthrosis	stiffness
6) Viscera:	rupture bladder	rupture urethra	others
7) Bone:	malunion	non union	osteomyelitis

N.B. Fractures common for nonunion:

Upper limb	Lower limb
1. - Fracture mid shaft humerus	1. -Fracture neck femur
2. - Fracture olecranon	2. -Fracture patella
3. - Fracture lower end ulna	3. -Fracture lower end tibia
4. - Fracture scaphoid	4. -Fracture medial malleolus

Clinical picture:

-History of trauma: (may be absent in pathological fracture).

-Pain & limitation of movement: (may be minimal in impacted fracture).

-Deformity: described according to the distal fragment (may be absent in undisplaced fracture).

-Tenderness & crepitus: Crepitus is painful, risky and may be absent over impacted fracture.

-Signs of complications.

Investigations

1-Plain X ray: *Indications* → Diagnosis of the fracture & displacement. / For medico-legal reasons. / To check reduction. *View* → AP / Lateral

2-CT scan: For certain fractures e.g. fracture pelvis & vertebrae.

Treatment:

Two Golden Rules

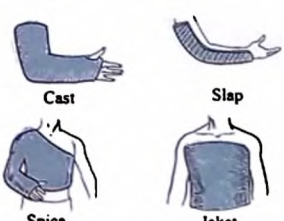
1- Save the patient's life first then save the patient limb.

2- Do not amputate life to preserve a limb.

I- First Aid:

1-Arrest hemorrhage and correct shock

2-Sound transportation: The fracture should be splinted.



II- Definitive management

A-Manage the life threatening conditions:

e.g. internal hemorrhage & visceral injuries.

B- Management of the fracture:

*1- **Reduction**:* Closed reduction or open reduction.

*2- **Fixation**:* In unstable fractures, the rule is to fix joint above and joint below the fracture.

-external fixation by plaster of Paris e.g. Casts, slabs, jackets,& spica.

-Internal fixation e.g. plate and screws & intramedullary nail.

*3- **Rehabilitation***: The joints outside the plaster should be moved from the first day, and those inside the plaster immediately after removal of the plaster.

Fractures of the upper limb

1- Fracture clavicle

Aetiology:

1- Indirect trauma: Fall on the outstretched hand.

2- Direct severe trauma: (rare).

Two contours & two curves

Pathology:

Types = sites.

1- Fracture middle 1/3 (80%):.It is the commonest site due to:

1- Junction of two contours.(triangular in the medial one third & flattened in the distal one)

2- Junction of two curves (convex in the medial one third & concave in the distal one) .

3- Further weakness by groove for subclavius muscle and foramen for a nutrient artery.

Displacement:

1- Proximal fragment: is elevated by sternomastoid muscle.

2-The distal fragment: moves downward, forward & medially by the weight of the upper limb & muscles of bicipital groove.

2- Fracture lateral 1/3 (15%):

Usually no displacement as the coraco-clavicular ligament fixes both fragments.

3- Fracture medial 1/3 (5%).

Complications: local

1-Skin: compound fracture is extremely rare although the clavicle is subcutaneous because:

a- The skin over is lax.

b- The strong sheet of platysma.

c- Direct trauma is a rare cause of fracture.

2-Vessels: injury to subclavian vessels (rare).

3-Nerves: injury to brachial plexuses (very rare).

4-Bone: Malunion with callus formation: of no functional disability but cosmetically bad (disappears in 1-2 years).

Deformity

Incidence:

It is the commonest long bone fracture in the body.

Clinical picture:

1- History of trauma (fall on outstretched hand) followed by pain and limitation of movement of the epsilateral UL.

2- *Deformity*: The displacement is usually apparent (subcutaneous bone) and the patient attains a characteristic position (like a mother feeding her baby) supporting his upper limb by the contralateral one (to prevent drop of the distal segment) and titling the head to the fracture side (to relax the sternomastoid muscle).

3- *Crepitus*: is very dangerous to be elicited.

Investigations:

Plain X-ray: is of diagnostic importance in.

1- Greenstick fracture.
2- Fracture lateral 1/3. } **Minimal displacement**

Figure of 8 bandage.

Treatment:

Previously: Closed reduction & external fixation

- While the patient is sitting on a low stool, the surgeon retracts patient's shoulders from behind using his knee as a fulcrum between the patient's scapulae to move the lateral segment upward, backward & laterally. Fixation is done by a *figure of 8 bandage* with a pad of cotton in both axillae.
- The bandage is tightened every 2 days for 2 weeks.

Recently: reduction is not advised (risky & unstable). Also, figure of 8 bandage is uncomfortable. Only, arm to neck sling for 3 weeks.

-Open reduction & internal fixation by kirschner wire in non-union or injury to vessels, nerves.

Arm to neck sling

Fractures of the humerus

I- Fracture upper end humerus.

Aetiology:

I- Direct trauma: Fall on the shoulder (anatomical neck & greater tuberosity).

II- Indirect trauma: Fall on the outstretched hand (surgical neck).

Pathology:

Types: according to *Neer's classification*:

The upper end of humerus is formed of 4 segments.

1- Articular segment (anatomical neck)
2- Greater tuberosity.
3- Lesser tuberosity.
4- Surgical neck.

Four segments

Fractures of the upper end are classified into:

1- *One part fracture*: fracture between any number of segments but with no displacement (the head remains one part).

2- *Two part fracture*: one segment is displaced (the head becomes 2 parts).

3- *Three part fracture*: two segments are displaced (the head becomes 3 parts).

4- *Four part fracture*: the four segments are displaced (the head becomes 4 parts).

Displacement:

The segment is considered displaced if there is > 1 cm. separation or > 45° angulation.

Complications:

1-*Nerve*: injury to the axillary nerve → Paralysis of the deltoid (flat shoulder deformity).

2-*Joint*: a- Dislocation of the shoulder.

b- Shoulder stiffness is common.

c- Haemoarthrosis.

3- *Bone*: a- Avascular necrosis of the head.

b- Malunion is common but doesn't interfere with function.

Incidence:

Surgical neck fracture is common in old age (osteoporosis).

Clinical picture:

- *History of trauma* (fall on the shoulder or the outstretched band) followed by *severe pain & limitation of movement* of the shoulder.
- *Deformity* may be present only in displaced fracture (20%).
- *Swelling* may present (haemoarthrosis).
- *Examine for possible complications* e.g. shoulder dislocation & axillary nerve injury.

Investigations:

Plain X ray: Indications ← Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction

View → AP; Lateral

Treatment:

I- One part fracture: (80%): Only arm sling until pain subsides.

II- Two part fracture:

1- Fracture lesser tuberosity: Only arm sling till pain subsides.

2- Fracture greater tuberosity: Closed reduction isn't satisfactory, because the rotator cuff muscles (supraspinatus, infraspinatus, teres minor & subscapularis) inserted into it usually cause re-displacement. So, internal fixation with screw or wire is required and the arm is put in arm sling.

3- Fracture surgical neck:

Old method: closed reduction and fixation of the limb in 30° abduction by putting cotton in the axilla and strapping the arm to the chest.

Recently collar and cuff allow gravity to reduce and fix it

4- Fracture anatomic neck: Avascular necrosis of the head is an imminent risk so; prosthetic arthroplasty gives the best results.

Collar & cuff

III- Three-part fracture:

Open reduction & internal fixation using wires or/and screws with repair of the rotator cuff

IV- Four part fracture:

There is high incidence of avascular necrosis, so, prosthetic arthroplasty with wire fixation of the tuberosities and repair of rotator cuff is advisable.

II- Fracture shaft humerus

Aetiology:

I- Direct trauma: usually causes transverse fracture.

II- Indirect trauma: → Fall on outstretched hand.

↘ Twisting trauma to the forearm → spiral fracture.

Pathology:

Types & displacement:

1-Just below the surgical neck.

Proximal fragment is abducted by supraspinatus.

Distal fragment is adducted by pectoralis major.

2-Above the deltoid tuberosity

Proximal fragment is adducted by pectoralis major

Distal fragment is abducted by the deltoid.

3-Below the deltoid tuberosity:

Proximal fragment is abducted by deltoid.

Distal fragment is elevated by biceps & triceps.

Just below the surgical neck

Above the deltoid tuberosity

Below the deltoid tuberosity

Complications:

1- Vessels: brachial artery injury (uncommon).

2- Nerves: - Radial nerve injury (not uncommon, 10%).

- Median nerve injury (rare).

3- Joint: stiffness of both shoulder & elbow.

4- Bone: malunion, delayed & nonunion due to injury to the nutrient art.

Clinical picture:

-*History of trauma*: (fall on outstretch hand or twisting trauma), followed by *severe pain and limitation of movement* of the limb.

-*Deformity*: may be obvious with true shortening of the arm (distance between the tip of acromion & lat. epicondyle is short).

-*Swelling*: may be marked.

-*Examine for* possible brachial artery or radial nerve injury.

Investigations:

Plain X-ray: *Value* → Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction. *View* → AP; Lateral

Treatment:

1- Closed reduction with external fixation:

Usually without anesthesia.

Reduction: by traction on the elbow with counter-traction by assistant hands in the axilla.

Fixation: by a U shaped plaster slab running from the acromion down to the lateral aspect of the arm then, below the elbow & then up the medial aspect of the arm to the axilla.

- Bandage is then applied to the arm to fix the slab. Finally apply a collar and cuff to maintain reduction by gravity traction for 6 weeks.

Rehabilitation:

-Movement of the hand and if possible the elbow from the start.

-From 3 to 6 weeks, the shoulder is mobilized outside the collar.

U shaped slab

II Open reduction & internal fixation:

When closed reduction fails, open reduction and internal fixation with plate & screw or intramedullary nail can be used.

III- Delayed union or nonunion

Internal fixation with bone graft.

III- Fracture lower end humerus

It includes:

1- Supracondylar fracture.

2- Intercondylar (Y or T fracture)

3- Condylar fracture (medial or lateral)

4- Articular surface: trochlea or capitulum.

5- Fracture epicondyle medial or lateral.

Supracondylar fracture of Humerus

Aetiology: Trauma

1- Fall on the outstretched hand (in children) → extension type.

2- Fall on the back of flexed elbow→ flexion type.

Extension Type — Flexion Type

Pathology:

Types:

1-Extension type: (99%)

Where the distal fragment is pushed backward & upward with pronation & tilt.

Grade I: Greenstick fracture with no or minimal displacement (angulation < 20 degree).

Grade II: Greenstick fracture with angulation > 20° but with posterior cortical continuity.

Grade III: marked displacement with no cortical continuity.

2-Flexion type :(1%)

Where the distal fragment is pushed forward and upward.

Complications: as usual but commonly:-

1-Vessels: Brachial artery injury (tear or more commonly compression). It may lead to acute ischaemia or subsequent Volkmann's ischaemic contracture .

2-Muscles: - myositis ossificans.

3-Nerves: - Median nerve injury: not uncommon.

-Radial nerve injury: rare.

-Ulnar nerve injury: late delayed ulnar neuritis due to cubitus valgus.

4-Joints: Stiffness of the elbow joint, due to myositis ossificans, malunion or adhesions.

5-Bone: Malunion: cubitus varus or valgus.

Incidence:

It is commoner in children. Similar trauma in adults causes post. elbow dislocation more likely

Clinical picture:

- *History of trauma* (fall on the ground) followed *by severe pain* and *limitation of movement* of the elbow with *swelling* in the distal arm.
- *Deformity* is usually marked with marked swelling and shortening of the upper arm.
- *Examine for* the vascularity of the limb & radial pulse cautiously.

Investigations:

1- Plain X ray: *Value* → To detect type & degree of the fracture; For medicolegal reasons; To check reduction. *View* → AP; Lateral

Differential diagnosis:

From posterior dislocation of the elbow:

Item	Supracondylar fracture	Post. dislocation of the elbow
1. Incidence	In children (common)	In adults (uncommon)
2. The relation between olecranon & the two epicondyles.	Preserved (straight line in extension & triangular in flexion	Disturbed
3. Movement	Limited	Lost
4. Measurements:		
Acromion to lat. epicondyle	Shortened	Normal
Lat. epicondyle to styloid process	Normal	Shortened

Treatment:

1-Grade I: It needs no reduction. Only fixation with a posterior plaster slab from the axillary fold to the knuckles of fingers with the elbow flexed then apply collar & cuff for 3 weeks

2-Grade II: Closed reduction & fixation.

Closed reduction is done by flexion of the distal fragment to correct the angulation then fixation as before.

3-Grade III: closed reduction is tried first. If failed, internal fixation is resorted to.

1-Closed reduction: is done urgently to relief compression on brachial artery. It is done by

a) Traction & counter traction.

b) Pushing the distal fragment anteriorly (in ext. type) or posteriorly (in flexion type)

c) Extension of the elbow.

d) Supination: to correct the pronation deformity.

e) Restore the carrying angle (the angle between the arm and the forearm. It is about 18°.

d) Flexion of the elbow slowly to 45° or to maximum degree of flexion compatible with presence of radial pulse. Fixation in posterior plaster slab is done as before

-The patient should be hospitalized for 24 hours during which the radial pulse is observed. If impaired → remove the slab & extend the limb. If it does not improve → explore the artery.

*Some surgeons prefer to fix both extension & flexion types in extension position because:

1-The risk of impeding the circulation to the limb is less.

2-Checking the carrying angle is better to avoid varus or valgus deformity.

3-Stiffness is rare in children.

2-Internal fixation: open reduction & internal fixation with kirschner wire is indicated if closed reduction failed.

Rehabilitation

-Exercise for fingers is started from the first day.

-The slab is removed after 3 weeks and replaced by sling for another 3 weeks during which active exercise of the elbow is started.

Volkmann's ischaemic contracture

Definition:

Fibrosis & shortening of the flexor muscles of the forearm due to their infarction secondary to brachial artery occlusion (compression or embolism).

Aetiology:

-Brachial artery injury in supracondylar fracture → infarction of flexors in the forearm → Contracture & fibrosis

-Ischaemia of the median & ulnar nerves adds more to atrophy and fibrosis.

Pathology:

-The flexors are shortened leading to flexion of the wrist & inter-phalangeal joints with hyperextension at the metacarpophalangeal joints (stretched extensors) →*claw hand deformity*.

-The median nerve being in the centre of the infarct muscles suffers more ischaemia than the ulnar nerve

Clinical picture:

1-Stage of acute ischaemia: 6 Ps

Pain, Puffness, Paraesthesia, Pallor, Paralysis & Pulesless limb & Cold fingers.

2-Stage of contracture:

-The muscles in the front of the forearm are atrophied.

-The wrist & inter-phalangeal joints are flexed & the metacarpophalangeal joints are extended.

-When the wrist is extended, the fingers become more flexed.

-Characteristically, all muscles have some function and not completely paralysed as in true nerve injury.

The deformity

Treatment:

I-Early cases:

Physiotherapy may stretch the fibrosed muscles.

II-Late cases: Many operations were described. The best is *muscle slide operation* in which, the common flexor tendon is detached from the medial epicondyle and fixed at a distal level

Myositis ossificans

Definition

It is a rare condition following trauma due to calcification & even ossification of an intramuscular haematoma.

Aetiology

Trauma causing haematoma & may be fracture. The detached periosteum may be driven into the haematoma causing ossification of haematoma

Pathology:

Sites

1-Brachialis muscle after supracondylar fracture.

2-Quadriceps muscles after blunt trauma to the thigh or fracture shaft femur.

3-Adductor muscles: after prolonged riding *"rider's bone or hunter's bone"*.

4-Rectus abdominis muscle: after rectus sheath haematoma.

*Gross pathology

The calcified haematoma forms a hard mass that limits movement of joints.

Clinical picture:

- Limitation of movement after removal of the slab.
- Hard swelling may be felt in the muscle (rare).

X-ray picture

Investigations

Plain X-ray: only after 4 weeks after trauma, there is faint calcification (cloudy area) in the region of muscles gradually becomes denser and even mature bone forms.

Treatment:

I-Prophylactic:

By early reduction and avoidance of massage & passive exercise.

II Treatment of established cases:

1-Early cases: rest the limb in cast again till pain disappears, then physiotherapy by graduated active exercise

2-Late cases: Excision of the ossified mass should be delayed for 6-12 months to avoid spread of ossification.

N.B. Myositis ossificans progressiva: congenital disease of unknown aetiology, characterized by progressive widespread ossification of the muscles and early death in childhood.

Fracture of the ulna

1) Fracture of olecranon:

See later with fracture patella.

2) Fracture of the shaft:

a) Isolated fracture:

There is no or slight displacement

b) Montaggia fracture dislocation:

Definition:

Fracture of upper 1/3 of the ulna with dislocation of the radial head due to tear in the annular lig

Aetiology:

1-Direct Trauma: to the back of the forearm (napout fracture) →anterior Monteggia (extension type)

2-Indirect trauma: fall on the outstretched hand → post Monteggia (flexion type).

Pathological Types:

-*Anterior Monteggia*: (extension type) 85%

Fracture of the upper 1/3 ulna with anterior angulation and anterior dislocation of the radial head.

Anterior Monteggia

-*Posterior Monteggia*, (flexion type) 15 %

Fracture of the upper 1/3 ulna with posterior angulation and posterior dislocation of the radial head.

Posterior Monteggia

Treatment:

1-Anterior Monteggia: (extension type)

- In children: Closed reduction (usually unsuccessful) and fixation in plaster cast in flexion for 2-3 months.
- In adults & if closed reduction failed in children → open reduction of the ulna usually with plate & screws. The head of the radius is reduced spontaneously

2-Posterior Monteggia: (flexion type)

- In children: Closed reduction (usually successful) and fixation in plaster cast in extension for 2-3 months.
- In adults & if closed reduction failed in children → open reduction usually with plate & screws. The head of the radius is reduced spontaneously.

3-Fracture of both bones upper limb

See later

Fractures of the Radius

1- Fracture head of the radius

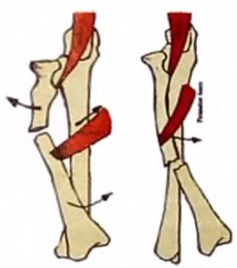
- It is caused by fall on outstretched hand
- In children it is in the form of slipped radial epiphysis which is treated by closed reduction or internal fixation
- In adults it is either fissure fracture which is treated by simple arm to neck sling or comminuted fracture which is treated by excision of the head of radius

2- Fracture shaft radius

Aetiology:

1-Direct trauma: common.

2-Indirect trauma: rare, due to fall on the outstretched hand.



above insert. of pronator teres | below insert. of pronator teres

Pathology

Types = levels

1-above insertion of pronator teres:

-*The proximal segment* is fully supinated by biceps & supinator.

-*The distal segment* is fully pronated by pronator teres & quadratus.

2-below the insertion of pronator teres

-*The proximal segment:* midway between supination & pronation.

-*The distal segment* is pronated by pronator quadratus.

N.B. overriding of both fragments doesn't occur in both types except if there is associated fracture of the ulna or dislocation of inferior radio-ulnar joint (Galeazzi).

Treatment

1-Closed reduction: is usually successful.

2-Fixation in:

-Full supination for the first level.

-Midposition (between supination & pronation) for the 2nd level in plaster cast for 3 months.

3- Galeazzi fracture dislocation

Definition: Fracture distal 1/3 of radius with dislocation of the inferior radioulanar joint.

Aetiology: Fall on the outstretched hand.

Treatment: Usually by open reduction & internal fixation.

Galeazzi fracture

3-Colle's fracture

Definition:

Fracture of the distal inch of the radius with or without fracture of the styloid process of the ulna

Aetiology:

Fall on the outstretched hand

Colle's fracture

Pathology:

•Displacement

-*The proximal segment* is fully pronated by both pronators (teres & quadratus), as they are stronger than supinator.

-*The distal segment* is displaced backward, upward & laterally

Complications: as usual but commonly

1-Bone:

- *Malunion*: If persistent posterior displacement, it leads to weak grip & pain.
- *Sudeck's atrophy* (post-traumatic osteodystrophy).

Definition: progressive decalcification of bone (the carpus) following trauma around wrist.

Aetiology: unknown, may be due to pain → sympathetic stimulation → hyperaemia → decalcification of bone (Garish law).

Clinical picture: - Severe pain, stiffness of the hand with some vasomotor disturbance e.g. coldness, cyanosis & oedema.

Investigations. X ray may show rarifaction of the carpal bones.

Treatment: -Immobilization in plaster.

-Novocaine block of the cervical sympathetic ganglia.

-Sympathectomy may be resorted to.

- *Fracture styloid process of ulna.*

2-Muscle: Fraying & rupture of the extensor pollicis longus tendon due to trauma (early) or due to friction against rough surface (late).

3-Nerve: Median nerve injury (rare).

4-Joint: -Stiffness of the wrist joint.

Dinner fork deformity

Incidence:

Common in old females (old women fracture) due to *osteoporosis*.

Clinical picture:

-*History of trauma* (fall on outstretched hand) followed by *pain and limitation of movement*.

-*Deformity:* The characteristic *dinner fork deformity* due to the backward, upward & lateral displacement of the distal segment.

-The two styloid processes may be felt in the same level (due to upward displacement of the radial one (normally, the radial styloid process is in a lower level than the ulnar one).

-The fracture is impacted →*no crepitus.*

-*Examine for* median nerve injury.

Investigations:

-Plain X Ray: *Value* ← Diagnosis of the fracture & displacement. / For medicolegal reasons. / To check reduction. *View* → AP / lateral

Treatment:

Recent cases

1-Closed reduction: by 3 grips technique.

First grip: shake hand with the patient with the other hand fixing the forearm. Then do traction with counter traction to correct the upward (impaction) deformity.

First grip

Second grip: posterior compression with pronation to correct the backward displacement & tilt

Third grip: lateral compression of the wrist to correct the lateral displacement & tilt

Second grip

2- Fixation:

- By below elbow plaster cast in pronation, mild flexion of the wrist & ulnar deviation for 6 weeks.

-If styloid process of the ulna is fractured, extend the cast above elbow.

Third grip

3- Rehabilitation:

Exercise of the fingers from the first day.

In neglected cases of mal-united cases, excision of the distal one inch of the ulna was done to relieve pain but the best is correction & internal fixation.

4-Smith's Fracture

-It is the reverse of Colle's fracture.

-It is due to fall on the dorsum of a palmar flexed wrist.

-The distal fragment is displaced forward & upward.

-Treated with closed reduction with above elbow cast for 6 weeks or internal fixation.

5- Fracture both bones of the upper limb

Definition:

It is fracture of both ulna and radius.

Aetiology:

1-Direct trauma: direct blow to the forearm → both bones are fractured at the same level.

2-Indirect trauma: A-Fall on the outstretched hand → fractures at different levels.

B-Torsion trauma to the forearm → spiral fracture of both bones

Pathology:

Displacement: It may be minimal as in greenstick fracture in children or with marked displacement (angulation, overriding & rotation). The radius shows rotation according to the level of fracture as in isolated shaft fracture (mention).

Complications = local

1-Vessels: rare.

2-Nerves: interosseus nerves (anterior & posterior), but uncommon.

3-Joints: dislocation & stiffness.

4-Bones: - Non-union (rare), malunion (common).

- Cross union (disabling) → loss of pronation & supination.

Clinical picture:

-*History of trauma*, followed by severe pain and limitation of movement.

-*Deformity* is usually obvious except in green stick fracture.

-*Swelling* is usually marked.

-*Examine for* vascularity & nerve injury.

Investigations

Plain x ray: *Value* ← Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction

View → AP; lateral

Treatment

1-Green stick fracture:

It is advisable to turn it complete fracture to gain some degree of over correction fixation is then done in above elbow cast with flexed elbow 90° and with neutral rotation for 6-9 weeks.

2-Displaced fracture:

A-Closed reduction & external fixation:

Closed reduction under anesthesia and fixation in above elbow cast with the elbow flexed 90°

-*Above insertion* of pronator teres→full supination.

-*Below insertion* of pronator quadratus → midway between supination & pronation.

-*Below insertion* of pronators quadratus → in full pronation.

B- Open reduction & internal fixation.

If closed reduction failed or redisplacement occured inside the cast, plates & screws are used for both bones. Each bone is explored through a separate incision to avoid cross-union

Rehabilitation:

-Exercise for the fingers from the start. Movement for the elbow is allowed after removal of the cast.

Plates & screws

Fractures of the vertebra

(Fracture spine)

Aetiology:

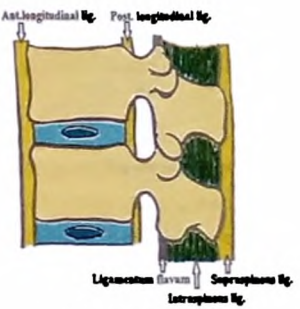
I- Direct trauma over the back

II- Indirect trauma: fall from a height and car motor accidents

a) Hyperflexion type (much commoner).

b) Hyperextension type (rare).

III- Pathological fracture: Wedging of vertebra under the effect of body weight in hyper-parathyroidism, osteoporosis … etc



Ligaments of the spines

Pathology

The stability of vertebrae is maintained by :

1-Ligaments:

a) Anterior & posterior longitudinal ligaments.

b) Interspinous & supraspinous ligaments.

c) Ligamentum flavum.

2-Intervertebral disc.

3-Interspinous joint: between articular processes of each neighboring vertebrae.

4-Paravertebral muscles.

Types:

Fracture spines are classified into stable & unstable according to the integrity of the poster ligament system (posterior longitudinal ligament + interspinous ligaments + supraspinc ligaments + ligamentum flava)

I-Stable Fracture Spine

-There is intact posterior ligament system

-There is no liability for further displacement or further cord injury

A-Wedge fracture:

Aetiology: hyperflexion trauma

Pathology:

-One vertebra collapses. It becomes wedge shaped.

-Intact posterior ligament system.

-No liability for spinal cord injury.

Wedge fracture

B-Comminuted (Burst) fracture:

Burst fracture

Aetiology: Fall from a height.

Pathology:

-The body of the vertebra is fractured & may be comminuted.

-The posterior ligament system is usually intact (no dislocation).

-The spinal cord or roots may be injured by displaced fragments of bone.

II Unstable fracture:

Fracture dislocation

Aetiology: flexion rotation trauma e.g. car accident.

Pathology:

-The body of the vertebra is fractured.

-The posterior ligament system ruptures (fracture-dislocation).

-The spinal cord or cauda equina may be injured or transected.

-The fracture is liable for further displacement & further injury to the cord.

Complications:

I-General complications:

-Shock. *-haemorrhage.* *-Embolism.* *-Ileus.*

-Complications of prolonged recumbency.

II Local complication:

Cord transection

1-Bone: malunion → kyphosis & scoliosis.

2-Nerves: cord or root compression, tear or transection leading to paraplegia or quadriplegia.

3-Joints: osteoarthrosis, stiffness.

Incidence:

- It is common in miners and building workers.
- It is more common in thoraco-lumber and cervico-thoracic region.

Clinical picture:

-History of trauma (severe trauma. but may be absent in pathological fracture), followed by *severe pain and inability to stand.*

-The deformity is usually not obvious, but in fracture dislocation, a gap can be felt between the spinous processes.

-Swelling is rare due to haematoma.

-Examine for:-internal hemorrhage and associated injuries.
-spinal cord or root injury.
-fracture calcaneus is a common association especially in fall down.

Investigations:

1-Plain X-ray: *Value* ← Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction — *View* → AP; lateral

2-CT. Scan: It is very important to detect the type of the fracture and exclude cord injury

Treatment.

A- First Aid:

1-Corrections of shock if present e.g. by blood transfusion.

2-Sound transport (very important): Patients with possible fracture spine should be transported as one block by three well trained persons to a hard porter with a supporting neck collar.

3-Internal hemorrhage & visceral injury have the priority to be managed first.

B- Definitive treatment:

I- Cervical spine fracture

1- Wedge fractures:

They are simply treated with immobilization in neck collar & analgesics.

2- Burst fracture:

Most of them should be considered unstable and they are treated by either:

a - immobilization and traction using skull calipers attached to a weight by a cord running over a pulley at the head of the bed. The bed is raised to about 45° to allow counter traction for several weeks. This is followed by fixation in *plaster jacket of Minerva* for three months.

b- Surgical reduction and fixation if the cord is at risk of injury.

3- Fracture dislocations:

They should be reduced surgically as early as possible with internal fixation using bone grafts.

II- Thoraco-lumbar fracture

1) Wedge fracture

-Rest in bed on a firm mattress till pain is relieved & Analgesic then active exercise in bed

-Weight bearing is allowed after one month in a special belt with iron bars in neutral position

2) Burst fracture:

-Fixation in plaster jacket to relieve pain and avoid malunion (e.g. kyphosis) for 3 months in neutral position (midway between flexion and extension)

- Weight bearing with the jacket is allowed after one month.

3) Fracture- dislocation:

A-Closed reduction & external fixation:

Closed reduction under morphia in hyperextension position using two tables with different height (*Watson, John's method*), then fixation in hyperextension for 3 months. Weight bearing is allowed after 1 month.

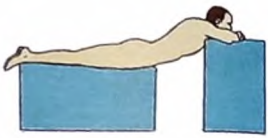
B-Open reduction & internal fixation:

Indications:

1-Difficult reduction (interlocking articular processes).

2-For decompression of the cord

3-For cases with paraplegia to facilitate nursing.



Watson John's method

Method:

-Open reduction may be with facetectomy

-Fixation with two plates fixed with screws to either side of the spinous processes of at least three vertebrae with the fractured one is the middle one

Rehabilitation:

-Back muscle exercise from the start. Weight bearing is allowed after one month.

Fractures of the pelvis

I-Fractures not involving the pelvic ring

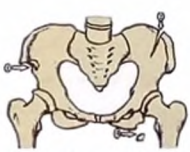
1-Avulsion fractures:

Aetiology: Strong muscle contraction.

Pathology: Types

Ant. Sup. Iliac Spine by powerful contraction of the sartorius.

Ant. Inf. Iliac Spine; by powerful contraction of the rectus femoris



Avulsion fractures & fracture of the iliac wing

2-Fracture of the iliac wing:

Aetiology: direct violence.

Pathology: No displacement as the iliac bone is supported by the iliacus muscle on its inner aspect & the glutei on its outer aspect.

Fr. Posterior margin

3-Fracture of the acetabulum:

Aetiology: indirect trauma transmitted through the femoral head

Pathology:

a) Fracture of the posterior margin in posterior dislocation hip.

b) Depressed or comminuted floor in central dislocation hip.

Comminuted Fracture

Fracture Acetabulum

4-Fracture of the sacrum:

Aetiology: -Direct trauma: fall or kick

-Indirect trauma: pelvic ring compression.

Pathology: It is usually transverse fracture at the lower border of the sacroiliac joint (undisplaced)

5-Fracture of the coccyx:

Aetiology: Direct trauma, fall or kick

Pathology: displacement is felt by PR.

Single fracture of the ring

II- Fracture involving the pelvic ring

1-Single fracture of the pelvic ring (incomplete type):

Aetiology: -Direct trauma: pelvic ring compression

-Indirect trauma: fall from a height

Pathology: The pelvic ring usually gives way at the weakest point (The two pubic rami).

Displacement: The pelvic ring is a strong osseoligamentous ring when broken at one site, no marked displacement occurs

2-Double fracture of the ring (complete type):

When the pelvic ring is fractured at two different places, this interferes with stable weight bearing & displacement may occur which may be difficult to reduce

a) Double fracture in the ant. segment of the pelvic ring:

Double fracture of the pubic rami usually with double fracture of the two ischial rami (*Butterfly fracture*). The central segment may be driven inward causing bladder or urethral injury.

Butterfly fracture

b) Open book fracture:

Aetiology: run over accident → pelvic ring compression.

Pathology: It consists of fracture of the pubic & ischial rami on one side (or separation of symphysis pubis) with iliac fracture (or dislocation of sacroiliac joint) .The pelvic ring opens up like a book

Open book fracture

c) Vertical shear fracture:

Aetiology: -Direct trauma: pelvic ring compression

-Indirect trauma: through the lower limb.

Pathology: The mobile hemipelvis is usually drawn up by the strong anterior & posterior muscle of the trunk

Vertical shear fracture

d) Lateral compression fracture:

Aetiology: Direct lateral trauma.

Pathology: The mobile hemipelvis is drawn inward to impact in the other hemipelvis.

Lat. compression fracture

N.B. disruption of the pelvic ring is caused by double fractures, double dislocations or fracture dislocation.

Complications:

I- General complications:

1- Shock: Neurogenic or hyporvolaemic.

2- Haemorrhage: Massive bleeding usually occurs in the last two types.

3- *Embolism*: Fat embolism.

4- *Ileus*: paralytic ileus from retroperitoneal haematoma or prolonged recumbancy.

5- *Complications of prolonged recumbency (*mention).

II- Local complications:

1- *Skin*: Compound fractures are rare with worse prognosis.

2- *Vessels*: great vessels (e.g. iliac vessels) may be injured.

3- *Nerves*: sacral nerves & lumbosaral plexus may be injured

4- *Viscera*: extraperitoneal rupture of the bladder & intrapelvic rupture of the urethra.

5- *Muscle*: severe muscle contusion & haematoma.

6- *Bone*: Malunion is of little functional importance but may interfere with normal labour in ♀

7- *Joint*: - Dislocation subluxation of the hip joint and osteoarthritis

Incidence:

It is more common nowadays in traffic accidents.

Clinical picture:

-History of trauma (severe) followed by severe pain and collapse with limitation of movement of the ipsilateral lower limb.

General:

- The patient may be shocked.
- Associated injuries are very common (polytrauma).

- Local:

- *Swelling* (haematoma or oedema)
- *Tenderness* -with distraction of the two ASISs
- Fracture coccyx or sacrum can be felt by PR. examination
- *Limitation of movement* of the ipsilateral LL.
- *Complications* may be evident e.g. rupture bladder or urethra. (discuss)

Investigations:

1-Plain X-ray: in A.P. lateral and oblique view.

2-U/S: to detect internal haemorrhage and organ injuries.

Treatment:

First Aid

- Stop haemorrhage & correct shock.

-Management of visceral injury if present e.g. rupture bladder or urethra (mention).

Definitive treatment

I-Fracture outside the pelvic ring

1-Avulsion fracture: bed rest for few days with the offending muscle relaxed.

2-Fracture of the iliac wing: firm bandage to the pelvis with bed rest till pain disappears.

3- Fracture of the acetabulum: -

1-*Linear fracture with no displacement*: skeletal traction for 6 weeks then no weight bears for another 6 weeks.

2-*Displaced fractures*: - open reduction & internal fixation.

4- Fracture of the sacrum: Reduction by PR digital manipulation then firm bandage.

5- Fracture of the coccyx: bed rest till pain subsides.

II-Fracture through the pelvic ring:

1-Single fracture of the ring: rest in bed till pain subsides.

2-Double fracture of the ring:

A-Butterfly fracture: uncomplicated fracture is treated simply by bed rest for 6 weeks (It is not in the pathway of weight bearing).

B-Open book fracture: reduction by lateral compression then fixation in hip spica or better external hip fixator.

C-Vertical shear fracture:

-Reduction & fixation by longitudinal skeletal traction.

-Open reduction & fixation may be indicated.

D-Lateral compression fracture:

-Minimally displaced fracture: bed rest for few days.

-Major displacement needs early reduction by traction & rotation of the femur then fixation by external fixator.

Hip spica

External hip fixator

Prognosis:

In the simple fractures, the mortality rate is about 8-15 %.

In the compound fractures, the mortality rate is about 50 %.

Fractures of the femur

1-Fracture of the neck of the femur

Aetiology:

1-In old age: usually minor trauma as when the foot catches the edge of a carpet. It is a pathological fracture and the patient falls as a result of fracture, not fractures because of fall.

2-In young age: longitudinal trauma along the femur

Pathology:

Types:

I-Intracapsular fractures:

1- Subcapital 2- Midcervical 3- Basal fracture.

II Extracapsular fracture (trochanteric fracture).

1- Pertrochanteric fracture.

2- Intertrochanteric fracture.

Displacement:

1-Adduction type (80%): the limb is flexed, adducted & externally rotated.

2-Abduction type (20%): the limb is flexed, abducted & externally rotated.

*Shortening occurs due to ascent of the greater trochanter by the pulling up of the glutei.

* *Pauwel's angle* is the angle between the fracture and the horizon. The narrower Pauwel's angle is, the better the impaction.

Pauwel's angle

Complications:

I-General complications:

Complications of prolonged recumbency.

II Local complications:

A-Bone:

1-Nonunion: due to

a) Avascular necrosis of the head (more in the intracapsular fracture) due to injury to the retinacular arteries to the head.

b) Instability due to the shearing effect of body weight.

c) Osteoporosis & osteomalacia.

d) Minimal haematoma (dry fracture): the synovial fluid causes lysis of the haematoma.

2-Malunion: Coxa vara & permanent shortening.

B-Joint: Osteoarthritis & stiffness of the hip joint.

Blood supply of the head

C-Nerves: Sciatic nerve injury is rare.

Incidence:

It is common in old age women with osteoporosis & osteomalacia.

Clinical picture:

-*History of trauma* may be absent or that of minimal trauma as stumble over the carpet or stairs followed by *pain & limitation of movement* at the hip joint. In impacted fracture, the patient may walk to the hospital but the patient cannot elevate the limb while the knee is extended.

-*Deformity:*

- External rotation causing foot to bed position. It is more in the extracapsular fractures.
- Shortening → short distance between ASIS & medial malleolus.

-The pulled up greater trochanter is diagnosed by:

Nelaton's line: while the patient is on his side, a line is drawn from the ASIS to the ischial tuberosity. Normally, it touches the tip of the greater trochanter. In fracture neck femur it passes through it

Nelaton's line

Investigations:

X ray: Impacted fractures with minimal displacement may be missed.

Treatment:

I-Intracapsular fractures.

A-Fit patient:

1. Patient under 65 years:

   Closed reduction followed by internal fixation with sliding compression cancellous screws: Two or three canulated cancellous screws are driven over guide wires. Bed rest for 2 weeks and walking on cruches for 4 months then weight bearing is allowed after radiological evidence of good healing

Cancellous screws

2. Patient over 65 years:

   The liability for avascular necrosis is high & the ability of the patient to withstand another operation is slight, so bipolar hemiarthroplasty is the treatment of choice (replacement of the head & neck of the femur) with bipolar hemiprosthesis.

Bipolar hemiarthroplasty

B-Unfit patients:

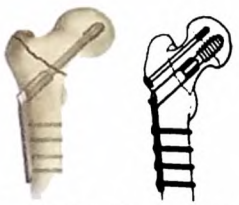
Rest in bed till pain subsides then moving on crutches for life.

II- Extracapsular fracture = Trochanteric fractures

A-Fit patients

Dynamic Hip Screw (DHS)

It is a half serrated nail introduced to fix the fracture then its backing out through the barrel of the plate will impact the fracture (lagging effect) with six hole plate to fix the trochanter and avoid fragmentation An anti-rotation screw may be added to avoid rotation of the head over the screw



Dynamic Hip Screw (DHS)

B- Unfit patients

Traction on Bohler's frame

III -Patients complicated with nonunion

The best management is total hip replacement.

2-Fractures of the shaft of the femur

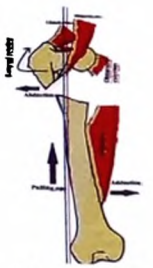
Aetiology:

1-Direct trauma:- It is common, which is usually violent trauma.

-Run over accident or fall from a height.

-Clumsy obstetric manipulation in breech presentation.

2-Indirect trauma (uncommon) e.g. falls from a height.



Subtrochanteric

Pathology:

Type & displacement:

1-Subtrochanteric fractures:

a) The proximal fragment is abducted by the glutei, flexed by the iliopsoas & externally rotated by obturator externus muscles.

b) The distal fragment is adducted & pulled up by the adductors.

Mid shaft

2-Middle two thirds fracture:

No special displacement but commonly:

a) The proximal fragment is adducted, flexed & externally rotated.

b) The distal fragment is pulled upward by quadriceps femoris muscle.

3-Supracondylar fracture:

a) The proximal fragment is flexed by iliopsoas & adducted by adductors

b) The distal fragment is sharply flexed backwards by the gastrocnemius.

Complications:

1-General complications:

1-Shock: Neurogenic or hypovolaemic.

2-Hage: The thigh may accommodate 2 liters of blood with intact skin.

3-Embolism: fat embolism (not rare).

4-Ileus: paralytic ileus.

5-Complications of prolonged recumbency (mention).

Supracondylar

II-Local complications:

1- Skin: Compound fractures carry a bad prognosis.

2- Muscle: Tear, gas gangrene, and myositis ossificans.

3- Vessels: Femoral artery & more commonly the popliteal artery in supracondylar fracture.

4- Nerves: sciatic nerve injury.

5- Joint: Haemoarthrosis or dislocation of the knee joint.

6- Bone: Malunion (shortening). -Non union (rare). - Osteomyelitis.

Incidence:

Common in civilian traffic accident.

Clinical picture:

-*History of trauma* followed by *severe pain & limitation of movement* of the affected LL.

-*Generally*: the patient is usually shocked.

-*Locally:*

* *Deformity* is usually apparent due to strong muscles. Angulation & shortening are marked.
* *Swelling* is usually marked due to haematoma.
* *Examine for* distal vascularity & other complications.

Investigations:

Plain X ray: *Value* → Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction. *View* → AP; lateral

Treatment:

I-First Aid:

-Correction of shock & treatment of life threatening conditions.

-Splint the fracture for sound transportation.

II- Definitive treatment of the fracture: <u>According to the age</u>

<u>1-New born</u> → *Crédé method:*

The fracture is splinted by two tongue depressors then the limb is splinted to the trunk.

Crédé method

<u>2-infant & young children < 5y:</u> *Bryant's method*:

By gallows traction for 1 month: Suspension of the child from his lower limbs on special gallows frame with the buttocks free of the bed to:

1- Perform traction on the muscles of the thigh.

2- Help easy cleaning & care of perineum.

3- Prevent pressure sores in the buttocks.

Gallows traction

<u>3-Older children (5-15y):</u>

By skin traction on *Thomas splint* as follows:

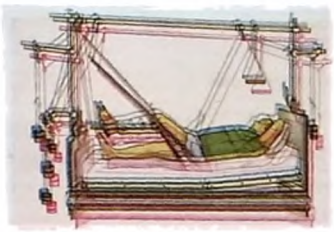
Skin traction on Thomas splint

- Reduce fracture under general anaesthesia.
- Apply adhesive plaster to both sides of the limb and apply bandage over the plaster.
- Tie the plaster tapes tightly to the end of the splint to perform traction. Counter traction is obtained by compression of the ring of the splint against the ischial tuberosity.
- This traction is maintained for 6 weeks replaced by hip spica for 6 weeks.

4-Adult above 15 years:

Either by a) skeletal traction.

Or b) Open reduction & internal fixation.



Skeletal traction on Balkan beam

A) Skeletal traction:

Indications:

1- Compound fracture.

2- Comminuted fracture.

3- Supracondylar fracture.

Method:

-Pass *Steinmann's pin* in the upper end of the tibia then connect it to a stirrup to which 5-7 kgs traction is applied through a cord passing over a pulley attached to *Balkan's beam.*

- Skeletal Traction is applied for 8 weeks, and then replaced by hip spica for another 8 weeks.

B-Open reduction & internal fixation:

Indications

1- Simple fracture.

2- Subtrochanteric fracture.

3- Supracondylar fracture & midshaft fracture.

4-Difficult closed reduction (soft tissue interposition).

5-Double level fractures.

6-Damage to vessels (firm stable fixation is mandatory).

Intramedullary & interlocking nails

Methods

1-Intramedullary & interlocking nail mainly in fracture middle 2/3 of the shaft.

2-Plate & screw well with dynamic compression plate mainly in subtrochanteric fr.

3-Condylar plate & screw in supracondylar fracture.

**Complications* of intramedullary nail:

1-Rotation if small nail is used (interlocking nail doesn't).

2-Impaction & fragmentation if large sized nail is used.

3- Migration in the gluteal region by early weight bearing

5-.Infection → osteomylitis 4-Fat embolism

6-Bending of the nail 7-It may stimulate bone growth in children.

**Contraindications of intramedullary nail: - 4 C*

1-Compound fracture 2-Comminuted fracture (not double level)

3-SupraCondylar. 4-Children.

Fracture patella or olecranon

Aetiology:

1-Direct trauma: falling on the knee or elbow.

2-Indirect trauma: Avulsion fractures.

- Forcible contraction of quadriceps e.g. falling from height.
- Forcible contraction of the triceps e.g. in electroconvulsive therapy.

Pathology:

Types:

1-Fissure fracture:- It is caused by mild direct trauma.

-It is linear with no displacement. It may be stellate shaped.

2-Avulsion fracture: -It is caused by indirect trauma.

- One pole is avulsed.

3-Transverse fracture: - It is caused by indirect trauma.

-Transverse fracture with wide separation.

4-Comminuted fracture: - It is caused by severe direct trauma.

-Multiple fragments with wide separation.

Complications:

1-Skin: Compound fracture is risky → septic arthritis.

2-Joint: (knee or elbow) -Haemoarthrosis. -Septic arthritis.

-Osteoarthrosis. -Stiffness.

3-Bone: - Nonunion: due to 1- Pull of quadriceps or triceps → imperfect fixation.

2- Soft tissue interposition.

-Malunion:→ osteoarthritis.

Incidence:

Fracture patella is common in middle age because avulsion trauma in old age usually ruptures the extensor expansion above the patella and in young age, it ruptures the ligamentum patellae below the patella. In middle age, it will fracture the patella.

Clinical picture:

-*History of trauma* (fall from a height on foot or on the knee or elbow) followed by *pain & limitation of movement* e.g. inability to extend the knee or the elbow actively .

-*Deformity:* a gap may be felt between the fragments of bone.

-*Swelling* is usually marked (Haemoarthrosis of knee or elbow)

Investigations:

Plain x ray: *Value* → Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction. *view* → AP; lateral

Treatment:

1-Fissure fracture or undisplaced fracture: → fixation

-Patella: - plaster cast for 6 weeks in extended position.

-Olecranon: - plaster cast for 6 weeks in flexion 90° (the functional position of the elbow).

2-Avulsion fracture: → partial excision.

-Patella: partial patellectomy & suturing the quadriceps tendon to the remaining part.

-Olecranon: excision of the detached part & suturing the triceps tendon to the remaining part.

3- Transverse fracture with displacement: internal fixation

- Patella: Open reduction & wiring (if perfectly reduced) and plaster cast for 6 weeks.
- Olecranon: open reduction and wiring or olecranon screw.

Screw

4- Comminuted fractures: excision

- Patella: total patellectomy and re-attaching the quadriceps -tendon to tibial tuberosity.
- Olecranon: excision of olecranon and re-attaching the triceps tendon to the head of ulna.

Wiring

Fractures of the tibia

Fracture both bones LL.

Aetiology:

1- Direct trauma:

-Road traffic accident: both bones are fractured at the same level.

-Compound fracture from without is common.

2- Indirect trauma:

a) Fall from a height:

-Fracture at two different levels (the lower 1/3 of the tibia & upper 1/3 of fibula)

-Compound fracture from within is common.

b) Twisting trauma: It causes spiral fractures.

Pathology:

Types: - The fracture may be:

- Transverse - Oblique - Spiral - Comminuted - Double level

Displacement:

The trauma is usually severe causing marked damage to bone & tissues with marked uncontrollable displacement.

Complications:

1- Skin: Compound fracture from without or from within.

2- Nerves: Lateral popliteal nerve injury.

3- Vessels: Popliteal, anterior tibial & posterior tibial arteries.

4- Joint: stiffness of knee & ankle.

5- Bone:

1-Non-union: common especially in the lower 1/3 tibia due to:

a- Injury to the nutrient artery.

b- Poor vascularity (the lower ½ of the tibia is surrounded by tendons not by muscles).

c- Soft tissue interposition.

d- High incidence of infection (compound fracture).

e- Major damage to bone & soft tissues (major trauma).

2- Malunion. 3-Cross union. 4- Osteomyelitis .

Incidence:

- It is common in traffic & war accident.
- It is common in athletes e.g. skiing, football.

Clinical picture:

- *History of trauma* followed by *severe pain and limitation of movement* of the lower limb.
- The patient may be shocked (multiple injured).
- *Deformity* is usually marked with skin wound. The bone ends may be visible in the wound.
- *Swelling* is usually obvious (deformity).
- *Examine for* the vascularity of the foot and other complications.

Investigations:

-Plain X ray: *Indications* → Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction. *View* → AP; lateral

Treatment:

I-First aid:

1- Correction of shock if present.

2- Compound fractures wound is cleaned with sterile solution and covered with sterile dressings.

3- If there is vascular injury, it should get the first attention

4- Splint the fractured limb during transport

II – Definitive ttt:

A- Simple fracture:

1- Transverse fracture: (stable reduction)

- Reduction under anesthesia.
- Fixation in plaster cast from the toes to above knee for 3-4 months.

2- Oblique & spiral fracture (unstable reduction).

- Closed reduction is unstable, so internal fixation with plate & screw better with dynamic compression plate is preferred.
- Intramedullary & interlocking nails may be used.

Internal fixation

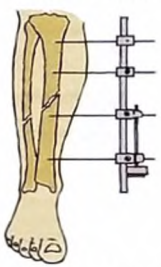
B – Compound fracture:

1- Compound from within (minimal contamination):

Manage as closed (simple) fracture and then suture the wound

2- Compound from without (marked contamination):

- Debride the wound and let it open.
- Reduce the fracture under anaesthesia.
- Fixation in plaster cast then a window is opened in the cast for dressing of the wound OR better external skeletal fixation leaving the wound exposed for dressing.
- For fractures with skin loss, skin grafts or flaps are used.



External skeletal fixator

C- Non-united fracture:

- Curettage of the bone ends + bone graft.
- Internal fixation better with compression palate.

D- Infected fracture:

1- Winette Orr technique + antibiotics.

2-Sequestrectomy (after complete separation) & saucerization.

External skeletal fixation

Pott's Fracture

Definition:

It includes fractures or fractures dislocation around the ankle.

Aetiology & Pathology:

Pott's fractures are classified according to the type of trauma into:

1- External rotation injury (the commonest):

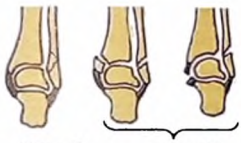
Aetiology: external rotation of the foot on the tibia (sudden arrest of the foot during walking)

Pathology: lateral twisting then medial traction trauma:

Stage I: spiral fracture of the lateral malleolus.

Stage II: stage I + rupture of the deltoid ligament or avulsion of the medial malleolus.

Stage III: stage II + fracture of the posterior margin of the tibia (the third malleolus).



Stage I stage II

External rotation injury

2- Inversion (adduction) injury:

Aetiology: hyper-adduction trauma.

Pathology: lateral traction then medial compression force.

Stage I: rupture of the lateral ligament or avulsion fracture of the lateral malleolus.

Stage II: stage I + vertical fracture of the medial malleolus

Stage III: stage II+ fracture of the posterior malleolus.

Stage I Stage II

Inversion (adduction) injury

3- Eversion (abduction) Injury:

Aetiology: hyper-abduction trauma.

Pathology: medial traction then lateral compression force.

Stage I: rupture of the deltoid lig or avulsion fracture of the medial malleolus.

Stage II: stage I + oblique fracture of the lateral malleolus.

Stage III: stage II + fracture of the posterior malleolus.

Stage I Stage II

Eversion (abduction) Injury

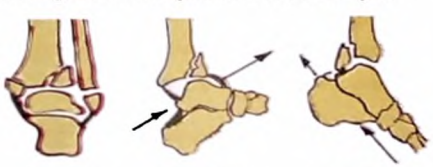
4-Central compression injury:

Aetiology: fall from a height.

Pathology: according to the position of foot during trauma. The pattern of fracture may be:

a) Burst fracture of the ankle:

If the foot was neutral at the time of trauma, the talus is driven up between the tibia & fibula with comminution of the lower end of the tibia.



Neutral dorsiflexed planter flexed

Central compression injury

b)- Anterior marginal fracture :-

- If the foot was dorsiflexed at the time of trauma, anterior subluxation of the talus occurs (fracture anterior margin of tibia).

c)- Posterior marginal fracture

-If the foot was planter flexed at the time of trauma, Posterior subluxation of the talus occurs (fracture posterior margin of the tibia).

5-Internal rotation Injury: (rare)

This trauma produces the same injury as those of adduction injury.

NB. Stage I fracture is considered stable fracture while stage II & III are unstable fractures.

Complications:

1-Bone: -Malunion.

-Non-union usually in fracture medial malleolus (soft tissue interposition).

- Sudek's atrophy of the metatarsals.

2- Joint: Osteoarthrosis of the ankle and stiffness.

Incidence:

Common in athletes (skiing)

Clinical picture:

- *History of trauma* followed by *severe pain & limitation of movement.*

-*Deformity* is usually evident.

- *Swelling* is marked.

Investigations

Plain X ray: *Value* ← Diagnosis of the fracture & displacement; For medicolegal reasons; To check reduction — View → AP; lateral

Stress X-ray: to detect ligamentous injuries.

Treatment:

I- Stable fractures.

Reduction under general anaesthesia and fixation with below knee plaster cast for 6 weeks.

II- Unstable fractures

1- Closed reduction & external fixation: Under general anaesthesia, correction of deformity then fixation in above knee plaster cast for 6 weeks. It is replaced by below knee cast for another 6 weeks then elastoplast for 4 weeks (the total period is 4 months).

2- Open reduction & internal fixation: It is indicated in:

1- If closed reduction fails. 2- In fracture medial malleolus.

Methods:

1- Screws. 2- Arthodesis in tibio-fibular diastasis with marked comminution.

March fracture

- It is crack fracture of the neck of the 2nd & the 3rd metatarsal bones that occurs during walking.
- It is due to repeated trauma of prolonged walking or military march so it is common in soldiers.
- It is treated by elastoplast bandage for 3 weeks.

Dislocations

It is a complete disruption of a joint so that there is no longer contact between the articular surfaces. If partial contact is still present, it is subluxation.

I- Shoulder dislocation.

Aetiology:

1- Indirect trauma:

1- Fall on the outstretched hand → - Behind the body → anterior dislocation.
- In front the body → posterior dislocation.
- Hyper-abducted → downward dislocation.

2- Over-extension, abduction & external rotation.

Pathology:

Types:

1- Anterior dislocation:

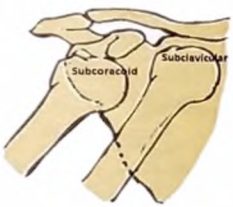
Subcoracoid and subclavicular type.

2- Posterior dislocation:

Subacromial and subspinous type.

3- Downward dislocation:

Subglenoid type (Luxatio en erecta).



Anterior dislocation

Posterior dislocation

Complications:

1- Vessels: injury of axillary vessels.

2- Nerve: injury of axillary nerve.

3- Bone: Fracture upper end humerus.

4- Joint: -Osteoarthritis, stiffness.

-Recurrent dislocation due to:-.

*injury to labrum glenoidale.

*rupture of the inferior part of the capsule.

Incidence:

-It is common in athletes.

-It is the commonest joint to dislocate because:

1- Large humeral head in relation to shallow glenoid cavity.

2- Wide range of movement.

3- Lax capsule & less supported by muscles inferiorly.

Luxatio en erecta

Clinical picture:

- *History of trauma* (fall on the ground) followed by *severe pain & loss of movement* at the shoulder joint.
- *Deformity:* loss of roundness of the shoulder → *flat shoulder deformity.*
- The head of humerus may be palpated in abnormal site.
  - *Hamilton's ruler test*: a long ruler can touch the acromial tip & lateral epicondyle at the same time (normally it can't).
  - *Duga's test:* - the patient can't touch the other shoulder while the elbow is in contact with the trunk.
- *Swelling*: due to haemoarthrosis, displaced head, and oedema.
- *Examine for*: axillary art & nerve injury.

Flat shoulder

Investigation:

Plain X ray: Indications → Diagnosis of the dislocation & displacement; For medicolegal reasons; To check reduction

View → AP; lateral

Treatment:

I- Recent dislocation: → *Kocher's method.*

Steady traction — External rotation — Hyperadduction — Internal rotation

Reduction:

1- Closed reduction: by steady traction on flexed elbow downward then,

2- External rotation to relax the subscapularis then,

3- Hyperadduction of the arm so that the elbow approximate the midline then,

4- Internal rotation of the arm so that the hand touches the opposite shoulder.

Fixation:

By strapping the arm to the trunk by adhesive plaster and neck to arm sling.

II- Neglected cases:

1- For few weeks: → *Hippocrate's method:*

While the patient lying on the ground the dislocation is reduced by strong traction against the bare foot of the surgeon in the patient's axilla then adduct the limb and fix it as before.

2- For few months: → Open reduction and external fixation.

3- Very old cases: may be left alone and they acquire a reasonable range of movement.

III Recurrent cases:

A- Conservative method: using check strap which is leather belt around the chest connected to another leather belt around the arm by strap that prevents wide movement.

Check strap

B- Operations:

1- *Odard's operation*:

Osteotomy of the coracoid process & its Depression in front of the shoulder joint to prevent dislocation.

2- Nicola's operation:

Division of the long head of biceps and passing it into a drill hole in the humeral head & sutured again to itself and to be intracapsular intrasynovial ligament.

Nicola's operation

3- Putti Platt's operation:

Division of the subscapularis muscle & the anterior capsule of the shoulder joint, then double-breasting them to limit ext. rotation.

4- Bankart's operation:

Surgical reattaching of labrum & the capsule to the glenoid cavity by silk sutures passing in drill holes in the bone.

Dislocation of the elbow

Aetiology:

1- Fall on the outstretched hand causing posterior dislocation.

2- Fall on the back of the elbow causing anterior dislocation.

Pathology:

Types:

1- Posterior dislocation: the ulna dislocates posteriorly commonly with fracture of the coronoid process of the ulna (the commonest type).

2- Anterior dislocation: the ulna dislocates anteriorly commonly with fracture of the olecranon.

3- Lateral and medial dislocations are rare.

Complications:

1- Bone: associated fracture of the olecranon or coronoid process.

2- Vessels & nerves: brachial artery & median nerve injury.

3- Skin: compound fracture dislocation with septic complication.

4- Muscles: myositis ossificans is not rare.

5- Joint: - Recurrence is not common.

- Osteoarthritis – septic arthritis – stiffness is common.

Incidence:-

It is common in children and young adults.

Clinical picture:-

- *History of trauma* followed by *severe pain and complete loss of movement of* the elbow.
- *Deformity*: -The olecranon is raised above the level of the two epicondyles.
- The distance between the lat. epicondyle & the styloid process of radius is reduced.
- *Examine for* complications e.g. ischaemia and nerve lesion.

Investigations:

- Plain X ray is diagnostic.

Differential diagnosis

From supracondylar fracture of the hurmerous (see before).

Treatment:

[1] Recent cases:

Closed reduction under anaesthesia and external fixation in plaster cast in 90 degrees flexion for one month.

[2] Old cases (unreduced for more than 1 month):

Open reduction and external fixation as before provided that the ends of bone are free and no myositis ossificans.

[3] Neglected cases (with degenerated ends of bone):

1) Arthroplasty: Excision of the lower end of the humerus, the head of the radius ,and the upper end of the ulna to enhance pseudo joint which is painless and mobile.

2) Sham reduction: in hopeless cases with myosltis ossificans. Under anaesthesia, the elbow is flexed so that the hand can reach the face (functioning position). The dislocation is not reduced and the joint is still immobile but in functioning position.

Hip dislocations

Aetiological types:

1- Congenital hip dislocation.
2- Traumatic.
3- Inflammatory: TB arthritis.
4- Neuromuscular: Poliomyelitis.
5- Neoplastic: metastases in the femoral head.

II- Traumatic dislocations

1- Posterior dislocation.

Aetiology:

1- Dash–board dislocation: when the dash–board strikes the knee of the front seated person especially when he puts one leg over the other (the position of flexion, adduction & internal rotation). In this case, any powerful force in the long axis of the femur pushes its head against the weak posterior part of the capsule.

Dash–board dislocation

2- Fall of roof dislocation: when a heavy object falls over the lower back of a stooping person with adducted lower limbs (LL).

Pathology:

Types:

1-Sciatic dislocation: the head is displaced directly backward to lie in greater sciatic notch.

2-Iliac or dorsal dislocation (commoner): the head is displaced backward & upward to lie in the back of the iliac bone.

Complications:

1-Bone: -Fracture posterior margin of the acetabulum.
-Fracture of the head of femur.
-Avascular necrosis of the femoral head.

2- Nerve: Sciatic nerve injury.

3- Muscle: Myositis ossificans.

4- joint: Osteoarthrosis of the hip.

Posterior dislocation

Incidence:

It is the commonest type of hip dislocation. It is common in traffic road accident

Clinical picture:

- *History of* severe trauma *followed by severe pain & loss of movement* at the hip joint.
- *Deformity:* Flexion, adduction & internal rotation.
- *Swelling:* the head of the femur may be felt in the gluteal region.
- *Examine for* sciatic nerve injury (drop foot).
- The femoral pulse is impalpable as the femur is displaced backward.

Investigations:

- Plain X ray: *Value* → Diagnosis of the dislocation & displacement; For medicolegal reasons; To check reduction. *View* → AP, lateral

Treatment:

-*Closed reduction:* by *Allis method*:

While the patient lying supine on the floor, an assistant fixes his pelvis while the surgeon flexes the hip & the knee 90° to bring the femoral head below the acetabulum then he lifts the femur vertically upward to reduce the head into the acetabulum.

Allis method

-*Fixation:* in hip spica or skin traction for 6 weeks in abduction.

Rehabilitation:

- Exercise for the toes & foot from the start.
- Exercise for the hip & knee after removal of the spica or traction.

2- Anterior hip dislocation.

Aetiology:

1- Hyper-abduction trauma: like when a person puts one leg on a boat leaving the bank while his other leg is still on the bank (*River bank dislocation*).

2- Direct trauma: a heavy trauma to the posterio-lateral aspect of one buttock.

Pathology:

Types and displacements:

1- Obturator type: the head dislocates anteriorly & inferiorly to lie on the obturator externus muscle.

2- Pubic type: the head dislocates anteriorly & superiorly to lie on the superior pubic ramus.

obturator type **pubic type**

In both types, the limb is flexed, abducted & externally rotated.

Clinical picture:

- *History of trauma* followed by *severe pain and loss of movement of* the hip joint.
- Deformity in the from of flexion, abduction & external rotation.

* In the pubic type, the limb is shortened but in the obturator type, the limb is lengthened.

- *Swelling:* the femoral head is palpable anteriorly with prominent femoral pulse.

Investigations:

- Plain X ray: *Value* → Diagnosis of the dislocation & displacement / For medicolegal reasons / To check after reduction
  View → AP / lateral

Treatment:

- Reduction is carried out by *(Bigelow's circumduction method)*:

Through exaggeration of flexion & abduction, then the thigh is rolled (circumfused) into adduction, extension & internal rotation.

* Care should be done not to fracture the femoral neck during reduction.

- Fixation: by tying the legs together for 3 weeks.

3- Central hip dislocation.

Aetiology:

1- Direct trauma: direct lateral trauma to the greater trochanter.

2- Indirect trauma: along the axis of the abducted femur.

Central dislocation

Pathology:

The head of the femur is forced inwards comminuting the floor of the acetabulum.

Clinical picture & investigations:

As usual

Treatment:

1- Reduction by bidirectional skeletal traction. The first is axial traction along the femur and the second is transverse through lateral traction on the greater trochanter for about 8 weeks.

2- Open reduction & internal fixation gives the best results.

III – Inflammatory conditions

Acute osteomyelitis

1- Acute haematogenous osteomyelitis.

2- Acute traumatic osteomyelitis.

3- Acute osteomyelitis due to extension from nearby infection e.g. osteomyelitis of the mandible secondary to dental caries.

1- Acute haematogenous osteomyelitis

Aetiology:

I- Causative organisms:

- Staph. Aureus 80 %.
- Other Gram positive bacteria: e.g. streptococci pyogenes, strept. pneumoniae.
- Gram negative bacteria e.g. E. coli, pseudomonas, proteus, H. influenza.

II- Route of infection:

Blood spread from septic focus commonly skin boil.

III- Predisposing factors:

1- Presence of septic focus.

2- Trauma: possibly causing small haematoma (good media).

3- Low body resistance: malnourished children.

Pathology:

Site: -In children, the commonest site is the metaphysis of long bones due to:

1- It is the most vascular part being newly formed immature bone.

2- It is the site of maximum stress due to attachment of tendons & ligaments.

3- The circulation inside it is slow being sinusoidal in nature.

- In adults, the vertebrae are a common site.

Pathological changes:

1- Inflammation of the metaphysis:

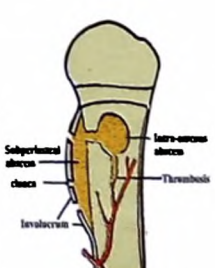
Local inflammation causes intraosseous abscess (in the third day) with marked rise of intraosseus pressure.

2- Extension & bone necrosis:

-Pus will spread in different directions:

a- To the medulla causing thrombosis of the nutrient artery.

b-Through the cortex to form subperiosteal abscess that raises the periosteum and obliterates the periosteal vessels.



Acute osteomyelitis

c- Through the epiphysis to the joint which is rare.

-Bone necrosis occurs due to obliteration of both medullary & periosteal blood supply to the cortex → *sequestrum* (separable dead fragment of bone).

3-New bone formation: in two places:

a- Under the lifted periosteum → *involucrum* which is pierced later by pus at multiple holes called cloaca, and will drains to the skin surface through sinuses. The condition now is an established chronic osteomyelitis.

b- Around Haversian canals in the surrounding bone → bone sclerosis.

4- Resolution & remolding:

When the condition is properly treated, healing occurs with fibrosis & bone sclerosis. Remodeling may restore the normal contour or fails & the bone is left permanently deformed.

Complications:

General:

Toxemia, septicemia & pyaemic abscesses.

Local:

1-Chronicity: → chronic osteomyelitis.

2-Pathological fracture.

3-Septic arthritis: rare because avascular epiphyseal plate will resist spread of infection except:

- In infant: still vascular epiphyseal plate.
- In adult: Fused epiphyseal plate.
- Congenital syphilis: Damaged epiphyseal plate.
- Strept. osteomyelitis: Its enzymes destroy the epiphyseal plate.
- Intracapsular metaphysis: e.g. hip joint.
- Intracapsular tendon: infection spreads around the long head of biceps to shoulder joint.

Incidence:

Common in children because:

1- Their metaphyses are growing & still vascular.

2- They are more liable to repeated trauma.

3- They are more liable to debilitating diseases.

Clinical picture:

Symptoms :

General: -Fever (even rigor) and other constitutional manifestations.

Local: - Pain: which is severe and

increases with	-dependency	*& decreases with*	-elevation
	-warmth		-undressing
	-movement		-Immobility

So, a child with acute osteomyelitis in his humerus tends to hang his arm up, bared & immobile (supported by the other limb).

Signs:

1- Inspection: -Hotness, redness, & oedema if the bone is subcutaneous.

-Oedema becomes pitting if subperiosteal abscess forms.

2- Palpation: -Tenderness over the affected bone maximum over the metaphysis.

-Sympathetic effusion in the nearby joint.

3- Percussion: Tenderness over the affected bone.

Investigations:

1- Leucocytosis.

3- X-ray: It shows no changes in the 1st 3 weeks.

Differential diagnosis:

1- Septic arthritis: -Absolute limitation of movement.

-Tenderness & swelling are severe over the joint.

2- Cellulitis: -Percussion of bone away from inflammation is not tender.

-Hotness, redness, oedema are more marked.

3-Ewing's tumour & osteosarcoma: -Toxemia is not marked.
-X ray is helpful
- Biopsy is indicated in doubtful cases.

Treatment:

I- Conservative treatment:

A- Rest: The rule is to put the patient in the bed and put the limb in a splint.

B- Improve the general condition: Dieting, vitamins and tonics.

C- Antibiotics: Anti-staph or broad spectrum antibiotics, which should be continued for at least 4 weeks inspite of general & local improvement of symptoms.

These measures usually succeed to control infection in early cases.

II- Surgical treatment:

Indicated if medical ttt fails → pus was formed.

Technique: The subperiosteal abscess is drained and one or two drill holes are performed in the metaphysis to drain the intraosseus pus.

Chronic osteomyelitis

Chronic non specific osteomyelitis:

1- Ch. haematogenous O M.

2- Ch. exogenous O M.

3- Ch. Periostitis.

4- Sclerosing osteoperiostitis.

5 – Brodie's abscess.

Chronic specific osteomyelitis:

1- Typhoid O M.

2- TB osteomyelitis.

3- Syphilitic osteomyelitis.

1- Chronic haematogenous osteomyelitis

= Ch. O.M. with sinus = Ch. O.M. on top of acute O.M.

Aetiology:

Improperly treated acute haematogenous OM. (improper antibiotic therapy and/or improper drainage)

Pathology:

See acute O.M.

Complications:

1- Pathological fracture.

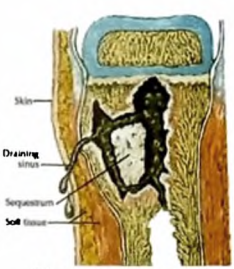
2- Disturbance of bone growth.

a- Deformed bone due to irregular healing.

b- Short bone due to damage of epiphyseal cartilage.

3- Amyloidosis: It may complicate long standing cases with large amount of pus.

4- Repeated acute exacerbations.



Chronic osteomyelitis

Clinical Picture:

History:

History of acute osteomyelitis and may be previous remission & exacerbations.

Symptoms:

Sinus or sinuses discharging pus and may be small fragment of bone (parts of sequestrum).

Signs:

- Multiple sinuses with protruding granulation tissue.
- The affected bone is thickened & tender.
- Probing of the sinuses leads to bone with metallic sound (sequestrum is devoid of periosteum). When sequestrum is completely separated, it moves with the probe.

Investigations:

1- Plain X ray:

-The affected bone is thickened & sclerosed.

- *Sequestrum*: dense opaque irregular sharply demarcated edge surrounded by a radiotranslucent zone when completely separated.

-*Involucrum*: irregular new bone formation around a cavity.

2- Radioisotope scanning: using ^{99m} Tc- HDP.

3- CT and MRI are important to show the extent of bone involvement and hidden foci of infection and plan for operative intervention.

X ray

Treatment:

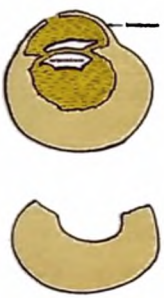
I- General measures:

Rest, antibiotics, and support of general condition.

II Surgical treatment:

-Wait until sequestrum separates, then do *Saucerization* i.e. Deroofing of the cavity to leave a shallow crater (to heal from the bottom) + sequestrectomy (removal of the sequestrum) + Excision of the sinus track.

- Fill the cavity with Vaseline gauze, bulky dressing and a plaster cast changed every 3-4 weeks. (*Winnet Orr technique)*]



Saucerization

2- Chronic exogenous osteomyelitis

= Chronic septic osteomyelitis

Aetiology:

Secondary to wound infection in:

a) Compound fracture. b) Amputation. c) Orthopedic operations.

Pathology:

- Pus is formed but drains through the wound (not under tension), so toxaemia & pain are less.
- Bone necrosis is limited to the ends forming an annular sequestrum.
- Bone formation only round Haversian canal → bone sclerosis.

Clinical picture:

- Picture of the original condition e.g. compound fracture.
- The wound discharges pus.
- Pain & toxaemia are less common.

Treatment:

1- Wide drainage of the wound.

2- Sequestectomy.

3- Winnet orr technique.

3- Chronic periostitis

Aetiology:

Infection of small subperiosteal haematoma.

Pathology:

- Periosteal irritation → excess new bone formation.
- Small cavitation may occur but sequestration is very rare and superficial.

Treatment:

- General measures. - Deroofing of the cavities. - Sequestrectomy (if present).

4- Sclerosing osteoperiostitis of Garré

Aetiology

Infection of long bone diaphysis with low virulent organism e.g. staph. albus

Pathology:

Nonsuppurative inflammation characterized by:-

- Excess subperiosteal new bone formation that will encroach upon the medullary cavity & around Haversian canals causing bone sclerosis.
- No cavitation or sequestration.

So the affected bone is thickened, sclerosed with narrow or obliterated medullary canal.

Sclerosing osteoperiostitis

Clinical picture:

Pain: dull aching, or severe pain due to increased intramedullary pressure. It is increased & decreased by the same factors as in acute osteomyelitis.

Investigations:

1- Plain X ray

2- Biopsy: to differentiate it from Ewing's sarcoma..

Treatment:

I- General measures.

II – Surgical treatment: Guttering of the whole affected area to relieve pain & let muscles inter the cavity increasing bl. supply & decreasing bone sclerosis.

Guttering

5- Brodie's abscess

Aetiology:

Infection of the metaphysis by low virulent organism e.g. staph albus in patient with good resistance (chronic from the start).

Pathology:

Infection causes a focus of inflammation, which is surrounded by a dense sclerosis that prevents spread of infection.

Site: metaphysis of long bones especially:

- The upper end of the tibia. - The lower end of the femur. - The upper end of the humerus.

Mac: Small cavity in the metaphysis filled with pus in the exacerbation or jelly like granulation tissue during remission and surrounded by a rim by of bone sclerosis.

Incidence:

Common in children & adolescents (of good resistance).

Brodie's abscess

Clinical picture:

- History of exacerbations & remissions.

-During attacks: pain, hotness, redness, oedema & nearby effusion.

Investigations:

Plain X-ray: radiotranslucent area surrounded with bone sclerosis.

Differential diagnosis:

I- From other bone abscesses:

1- TB. cold abscess = localized encysted TB osteomyelitis.

-It is epiphyseal. - No bone sclerosis. - The joint is usually affected.

2- Typhoid abscess:

- No bone sclerosis. - It contains chocolate pus. - Response to chloramphenicol.

3- Syphilitic abscess:

- Excessive scleroses - No tenderness - Other criteria of $

II From osteoid osteoma:

- It affects the shaft - characteristic pain - may be nidus in x ray.

Treatment:

I- General measures:

II–Surgical ttt: Saucerization, scrapping of the pyogenic membrane. Care should be taken not to injure the epiphyseal plate. Then Winnett Orr technique.

6- Typhoid osteomylitis

Aetiology:

Causative organism: Typhoid bacili (salmonella typhi).

Route of infection: bl. born.

Pathology:

Site: common in the upper end of the tibia, ribs, sternum & spines.

The abscess cavity is filled with granulation tissue & red brownish pus called chocolate pus.

Clinical picture:

- History of typhoid fever usually months or even years before but it may occur during the course of the disease.
- Pain,& tenderness, over the affected bone.

Investigations

1- Widal test: It is usually negative.

2- X-ray: bone cavity with no bone sclerosis.

Treatment:

1- Medical ttt: anti-typhoid therapy (usually effective).

2-Surgical ttt: 1-Unimportant bone e.g. ribs → excision.

2- Important bone e.g. tibia → saucerization & curettage.

7- TB osteomyelitis

TB of the spine = Pott's disease of the spine

Aetiology:

Causative organism: Mycobacterium tuberculosis.

Route of infection:

1-Blood borne: the commonest

2-Lymphatic borne : through cisterna chyli & thoracic duct.

3-Direct spread from lung or pleura.

Predisposing factor: low resistance of the patient.

Pathology:

Site:

The disease affects the vertebrae under stress e.g. vertebrae at the junction of mobile & fixed or less mobile vertebrae & vertebrae of greatest mobility so, the dorsolumber, lumbosacral & cervicodorsal regions are the commonest sites in this order of frequency.

Pathological changes:

1-In children:

The initial focus is the center of two adjoining vertebrae due to the peculiar blood supply of the vertebrae. TB. osteomyelitis leads to damage of the vertebral bodies and the intervertebral disc inbetween with collapse & deformity of the vertebrae. Cord compression is rare.

II- In Adults:

The initial focus is subperiosteal → T.B periostitis → cold abscess under the anterior longitudinal ligament which may pass into different directions according to the site of the 1ry disease.

BL supply of the spine

A -In the cervical region:

1-It may protrude anteriorly to form a retropharyngeal abscess.

2- It may protrude laterally to appear in the posterior triangle of the neck and may pass with the cords of brachial plexus to the axilla ,elbow & wrist.

B- In thoracic region:

1-It may protrude laterally → paravertebral abscess that may pass with the intercostal nerves to point with its lateral or its anterior cutaneous branch to the skin.

2-It may pass anteriorly to form a mediastinal abscesses.

C- In the lumber region:

It may pass laterally to form paravertebral, lumber or psoas abscess. The latter may pass with the psoas to point below the inguinal ligament in the thigh.

N.B. Cold abscess may compress the cord causing paraplegia.

Complications:

I- General complications:

1- T.B. Toxaemia 2- Miliary T.B. 3- Amyloidosis.

II- Local complications:

A- Paraplegia: it may be early or late.

1) Early paraplegia due to:

a) Compression by psoas abscess.

b) Compression by sequestrated piece of bone.

c) T.B meningitis & myelitis.

2) Late paraplegia (many years later) due to friction of the cord against the hard sharp gibbous (angulation).

Psoas abscess

B- Cold abscess:

-More common in adult.

-It may point elsewhere (see before).

C- Deformity: In the form of kyphosis, scoliosis or kyphoscoliosis.

Incidence:

Common in males and children.

Clinical picture:

I- Uncomplicated cases.

1- Pain:

-*Site*: the affected vertebrae. -*Radiation*: along the nerve roots (DD acute abd.).

-*Character*: dull aching. -*Intensity*: moderate to severe.

-*Increased by*: movement. -*Decreased by*: rest & immobility.

2- Localized tenderness:

Through pressure or percussion of the affected vertebrae.

3- Rigidity & limitation of movement:

-The patient avoids movement of the affected vertebrae & the paravertebral muscles are spastic.

a- In cervical Pott's disease:

- The patient supports his head by both hands.
- He turns the whole trunk to look to one side.

b- In dorsolumber Pott's disease:

- The patient limps due to psoas spasm.
- On picking up something from the ground, the patient squats with his back stiff and on getting up he climbs his lower limbs *(Coin test)*.
- The patient can't jump from a low stool *(stool test)*.

II- Complicated cases

1- Paraplegia:

- It firstly affects motor then sensory and lastly sphincteric function.
- Lower motor neuron lesion at the level and upper motor neuron lesion below the level.

2- Cold abscess:

- It points elsewhere as a large soft cystic painless swelling.
- Psoas abscess gives large cystic swelling that may point in the femoral triangle with cross-fluctuation between the abdominal & femoral parts.

3- Deformity:

In the form of kyphosis, scoliosis or kyphoscoliosis.

Investigations:

1- Pain X-ray: of the spine may show.

a- Destruction of at least two adjoining vertebrae with their intervertebral disc.

b- Soft tissue shadow of paravertebral or psoas abscess .

c- Crowdness of the ribs on the affected side (scoliosis) & kyphosis

2- Tuberculin test, ESR & blood picture.

3- Aspiration of accessible cold abscess for bacteriological exam.

Radiological findings

Treatment:

I- Uncomplicated cases

I- Conservative treatment:

A- General:

Sanatorium, diet, & antituberculous drugs.

B- Local:

Immobilization: -

1- Special frame in bed till quiescence of the disease which is proved:

- Clinically by decrease of toxaemia, pain & tenderness.
- Laboratory by falling of ESR.
- Radiologically by evidence of calcification.

2- Then plaster jacket till evidence of healing.

3- Special spinal brace for 1-2 years.

* Patient is observed in this period for signs of reactivity if they reappear→ re-immobilization

Posterior spinal fusion

The aim of immobilization is to

a) limit bone destruction & deformity.

b) give rest and chance for healing.

c) reduce pain and relieve spasm.

II- Surgical treatment:

Aim: To fix the spine to avoid deformity and cord compression.

Procedure: spinal fusion operation.

1-Posterior spinal fusion: through post. approach (extra-articular arthodesis using tibial graft).

2-Debridment-fusion operation: Preferably through ant. approach (thoractomy or laparotomy). The dead bone, disc material and caseous material are removed and bone grafts are put and the wound is closed.

II- Complicated cases

a) Paraplegia:

Decompression of the cord. The best decompression is *lateral rachiotomy* i.e removal of • the transverse process of the vertebra, • the posterior part of the rib, • the pedicle & • the posteriolateral part of vertebra.

N.B. Laminectomy is not preferred as it is a bad drainage operation and furtherly weakens the spine.

Lateral rachiotomy

2) Irreversible paraplegia: Nursing care.

B- Cold abscess:

Paravertebral cold abscess → drainage operation → *costotransversectomy* i.e removal of the transverse process with the posterior part of the rib.

Costotransversectomy

C- Deformity: e.g. kyphosis or scoliosis:

1) Early in children: it is correctable by positioning in bed.

2) Late in adult: it is uncorrectable → physiotherapy.

Prognosis:

- The mortality rate in Pott's disease is 10 %.

Osteochondiritis juvenilis

Definition:

It is a group of self limiting diseases of epiphyses characterized by compression, fragmentation or separation of the epiphysis.

Legg-perthes disease

Definition:

Osteochondritis juvenilis of the hip.

Aetiology:

Minor trauma → hip effusion → compression of retinacular aa → avascular necrosis of the head.

Pathology:

It passes through the following stages.

1- *Ischaemia & bone death*: Avascular necrosis of the head.

2- *Softening & fragmentation:* the femoral head flattens (coxa plana) and breaks into fragments. The metaphysis is hyperaemic (rarified) .

3- *Healing & reconstruction:* The bony architecture is restored but usually the femoral head remains deformed.

Incidence:

- It is common between 3-10 years.
- It is more common in ♂ than ♀.

Clinical picture:

- History of trauma in 50 % of cases.
- Pain in the hip referred to the knee with limping & mild limitation of movement.

Investigations:

Plain X ray: the femoral head appears more dense then flattened *(mushroom appearance)*.It then becomes fragmented followed by restoration of normal density.

Treatment:

Skin traction till pain is relieved then mobility is allowed without weight bearing by:

-using crutches.

-using special appliances that transmit the weight from ischium to the ground.

IV-Neoplasia

I- Benign tumours

A- Osteoma

1- Ivory (compact) osteoma.

Origin: from membranous bone especially the skull (outer or inner table).

Pathology: very dense solid mass fromed of compact bone.

Complications:

1- Cosmetic appearance.

2- When it grows from the inner table, it may compress the brain.

- In the orbit → proptosis.
- In the nose → nasal sinusitis.
- In the ear → deafness & vertigo.

Ivory osteoma

Clinical picture

It is that of complications.

Treatment:

1- If small → excision by trephining the tumour.

2- If large → drill holes around the mass then sawing with Gigli saw.

2-Cancellous osteoma = osteochondroma = exostosis

Origin: metaphysis of long bones especially lower end femur, upper end tibia & upper end humerus.

Pathology

-Hook shaped mass arising from the metaphysis and directed away from the joint.

-It is composed of pedicle of normal bone (cortex & cancellous bone) covered with a cap of cartilage with adventitious bursa.

-The tumour continues to grow till the normal time of epiphyseal fusion when the cartilaginous cap ossifies.

Osteochondroma

Types:

1- Single osteochondroma.

2- Multiple osteochondromata = ***multiple exostosis*** = ***<u>metaphyseal aclasis</u>*** where there are

- Short stature. - Broad metaphysis. - Multiple osteochondromata.

Complications:

1- Pressure on nerves or tendons.

2- Mechanical blocking to a joint movement.

3- Adventitious bursitis.

4- Malignancy – chondrosarcoma (5%).

Metaphyseal aclasis

Incidence:

It is more in children & adolescents.

Investigations:

Plain X-ray: shows the tumour.

Treatment:

1-Solitary: complete excision & biopsy.

2-Multiple: complete excision & biopsy of the complicated one.

Osteoid osteoma

3-Osteoid osteoma:

Origin: shafts of long bones e.g. tibia & femur.

Pathology: small (< 1 cm) reddish, brown, & firm nodule.

Incidence: It is common between 10 –25 years.

Clinical picture

-Pain which increases at night & is relieved by salicylates.

-Localized bone tenderness.

-There may be palpable bone thickening.

Investigations:

Plain X ray: radiotranslucent area surrounded by bone sclerosis and may contains small osseous shadow called nidus (new bone formation).

Treatment:

Complete excision with small area of the surrounding bone & biopsy after precise localization.

B-Chondroma.

1-Solitary chondroma:

Origin flat bones e.g. ribs, scapula, sternum, and pelvic bones.

Pathology lobulated mass of cartilage. It may grow centrally (enchondroma) or eccentrically (ecchondroma).

Complications:

1- Pathological fracture.

2- Malignant transformation → chondrosarcoma (1%) especially ecchondroma.

2-Multiple chondromata (Ollier's disease):

Chondromata

Origin: tubular bones of the hand & feet.

Pathology: masses of unossified cartilage.

Complications: as before +

1- Retardation of growth of the bone.

2- Deformity.

Clinical picture of chondroma:

1-Mass: expanding the finger.

2-Pain due to stretch of the periosteum.

3-Picture of complications.

Investigations

Plain X ray: well defined radio-lucent shadow.

Enchondroma: → thinning out of the cortex.

Ecchondroma → eccentric growth from the cortex.

Differential diagnosis:

From T.B panostitis (dactylitis).

Treatment

Excision or better curettage and putting bone chips in the cavity.

II- Malignant tumors

Classification

A-Primary tumors:

I-Mesenchymal tumors	**II-Myelogenic tumors**
1-Osteosarcoma.	1-Ewing's sarcoma.
2-Osteoclastoma.	2-Multiple myeloma.
3-Chondrosarcoma.	3-Leukaemia.
4-Fibrosarcoma.	4-Reticulum cell sarcoma.

B-Secondaries in bone:

They are more common than primary ones.

1-Osteosarcoma

Aetiology:

I-Cells of origin: osteoblasts.

II-Predisposing factors:

a) Irradiation: It was common in watch dial painters.

b) Trauma.

III-Premalignant conditions: Paget's disease of bone.

Sites

Pathology:

Sites: metaphysis of long bones especially lower end femur, upper end tibia, and upper end humerus.

MAC: There are two types:

1-Osteoblastic (differentiated) type: gray in colour. The cut section shows cartilage & bone.

2-Osteolytic (undifferentiated) type: is reddish brown in colour and soft in consistency. The cut section shows extensive areas of haemorrhage & necrosis.

MIC:

1-Malignant cells: spindle shaped cells with criteria of malignancy

2-Matrix: variable according to the differentiation. It may be osseous, cartilaginous or fibrous.

3-Blood vessels: are invaded by malignant cells.

Spread:

I-Local:

1-Intrinsic:

*To medulla: early.

*To cortex: - It elevates the periosteum → new bone is formed:

- Around the lifted periosteal vessels.

- At the angle between periosteum &shaft.

* Not to epiphysis: epiphyseal cartilage is respected

Spread

2-Extrinsic: late, the tumour infiltrates the periosteum to the surrounding structures

II- lymphatic spread: to the draining L.N.

III- Blood spread: very early to the lung.

Staging:

T.N.M classification

T0: No detected 1ry.	N0 = No L.N.	M0 = No distant metastasis.
T1: It didn't infiltrate the cortex.	N1 = LN. metastasis.	M1 = Distant M.
T2: It infiltrated the cortex.		

Complications:

1-Distant spread: the cause of death.

2-Cachexia: from insomnia due to pain & toxaemia.

3-Pathological fracture: not common due to pain that restricts movement.

Incidence:

The rule of 80

-80% occurs between 10-20 years (20% > 50 y).

-80% occurs in metaphysis.

-80% occurs in the lower limb, of which 80% occurs around the knee joint of which 80% occurs in the lower end of the femur.

Clinical picture:

1-Pain: is early & severe simulating that of osteomyelitis.

2-Swelling: characterized by:

- It appears months after pain.
- Rapidly growing.
- Ill defined.
- warm with dilated veins & may be pulsating.
- Variable in consistency.

3-Pathological fracture: not common.

4-Late complications: anorexia, asthenia, anaemia & low grade fever.

Investigations:

1-Plain X ray:

A- Plain X-ray of the affected bone shows the following:

i. Eroded cortex that appears hazy within the tumour (*bone ghost*).
ii. Extension into the medulla.
iii. Epiphyseal cartilage is respected.
iv. New bone formation in:-
   - Inside the tumor.
   - At the angle between periosteum and the shaft → *Codman's triangle.*
   - Around the stretched periosteal vessels →*Sun rays appearance.*

X ray

B- *Plain X-ray chest*: to detect secondaries (not reliable).

2-CT, MRI & body scan: to detect spread.

3-Biopsy: should be done before radical resection.

Differential Diagnosis:

From acute osteomylitis: Less toxaemia & fever. Radiological findings and biopsy must be done in questionable cases.

Treatment:

1-Operable cases:

- Early wide resection is the only hope of cure.
- Amputation with safety margin (e.g. with joint above the tumour).

NB. -Single lung metastasis isn't a sign of inoperability and can be treated with lobectomy.

II- Inoperable cases:

- Signs of inoperability:
  1- Inaccessible tumor e.g. vertebra.
  2- Extension to vital organs e.g. aorta.
  3- Multiple lung secondaries.
- They are treated by radiotherapy, although radioresistant, to relieve pain. Morphia may be used.

Prognosis:

-Prognosis is bad due to early metastasis.

-The survival rate is 50 % after 1 year & 10-20 % after 5 years.

2-Osteoclastoma

=Giant cell tumour (GCT)

Cell of origins:

Most probably spindle cells (not osteoclast).

Pathology:

Sites: Epiphysis of long bones especially lower end femur, upper end tibia & fibula, upper end humerus, & lower end radius & ulna.

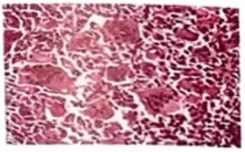
MAC: Soft friable reddish brown mass expanding the cortex. The cut section shows bone trabeculae inside a haemorrhagic mass.

MIC:

- *Two types of cells*
  a) Spindle shaped cells whose characters determine the behavior of the tumor, benign, invasive or frankly sarcomatous
  b) Giant multinucleated cells of osteoclastic type
- *Matrix :* vascular collagenous stroma

Sites

• *Areas of hemorrhage. & necrosis.*



MIC

- It was traditionally classified into three grades:

1- Grade I: benign giant cell tumor.

2- Grade II: locally malignant tumor.

3- Grade III: malignant or sarcomatous type.

Spread:

I-Local: 1- Intrinsic: It expands the cortex which is thinned out, but it doesn't spread:

* Through the cortex except in sarcomatous type.
* To the medulla due to operculum (cover) effect of medullary plug.
* To the joint (articular cartilage is respected).

2- Extrinsic: in sarcomatous type (malignant osteoclastoma).

II –Lymphatic spread:
III- Blood spread:
} Only in sarcomatous type

Complications:

1-Pathological fracture is common due to thinned cortex & late pain.

2-Recurrence: after curettage.

3-Distant metastases: to the lungs (rare).

Incidence: -

-It is common between 20-40 years (after union of epiphysis) with no sex predilection.

Clinical picture:

1-Swelling:

- It appears early before pain.
- Slowly growing.
- Well defined edge.
- Warm.
- Variable in consistency, but commonly it gives egg shell crackling sensation on pressure.

2-Pain: which is relatively late & mild.

3-Pathological fracture: is common (15%) as movement is not limited by pain.

4- Picture of complications.

Investigations:

1-Plain X ray of the tumour shows:

a. Marked expansion of the epiphysis with thinning out of the cortex. But no soft tissue shadow outside the cortex.

b. Bone destruction with no bone formation that leads to large cavities with pseudotrabeculae (remnants from bone) inside giving the typical *Soap bubble appearance.*

c. Medullary plug due to localized bone sclerosis = *operculum*

d. Articular cartilage is respected.

X ray picture

Differential Diagnosis

From other bone tumour especially osteosarcoma.

Treatment:

1-Curettage and filling the cavity with bone chips:

-It is indicated only in small slowly growing tumour.

-Recurrence rate is about50%.

II - Excision with safety margin:

-Unimportant bone: e.g. upper end of fibula, → excision without marked disability.

-Important bone, e.g. lower end femur, lower end radius → excision with reconstruction using:

A- Autograft e.g. upper end fibula.

B- Allograft: osteochondral graft from bone bank.

C- Prosthesis: partial or total knee replacement

III -Amputation is reserved only for

1-Malignant osteoclastoma.

2-Recurrence after curettage.

3-Chondrosarcoma

1-Primary chondrosarcoma:

- It occurs de novo (in normal bone).
- It simulates osteosarcoma in many aspects but differs in that:
  - * It occurs in older age group (40-60years).
  - *It tends to occur in flat bones, e.g. pelvic, scapula.
  - *It has a better prognosis.

2-Secondary chondrosarcoma:

- It occurs on top of metaphyseal aclasis or multiple chondromatosis.
- Malignant changes are suspected when:
  - * Pain occurs.
  - * The tumor grows rapidly.
  - * It recurs after local excision.
  - * The growth continues after union of epiphysis.
- It is common between 30 –50 years.

- It occurs in flat bones and metaphysis of long bones.
- It gives fluffy cotton appearance in X-ray.
- It has a relatively good prognosis (5years survival rate is about 50%).

4-Fibrosarcoma

-Site, clinical picture & spread like osteosarcoma.

-It is common in young adults of both sexes.

-It is an osteolytic tumour with high incidence of pathological fractures.

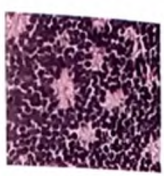
-It is treated with early amputation.

-The 5 year survival rate is about 30%.

5-Ewing's sarcoma

Cells of origin:

Most probably from vascular endothelium in the bone marrow.



MIC

Pathology:

Site: -Diaphysis of long bones especially the lower limb.

MAC: Soft grayish mass. The cut section shows areas of hage. & necrosis.

MIC: Rounded cells arranged in a rosette manner around the blood vessel.

Spread

I-Local:

1-Intrinsic:

-To the medulla early.

-To the cortex → It elevates the periosteum that reacts by new bone formation, which is furtherly invaded by malignant cells raising the periosteum again → new bone formation & the cycle is repeated→ *onion peel appearance.*

2-Extrinsic: late.

II- Lymphatic: to the draining LNs.

III- Blood spread: early to the lung and other tissues even to other bones.

Onion peel appearance

Complications:

1-Distant metastasis.

2-Pathological fracture. It is rare due to pain which limits the movement & the new bone formation by periosteum.

Incidence:

It is common in 5-15 years children.

Clinical Picture

1-Pain: which is early & severe simulating that of osteomyelitis.

2-Swelling: which is:

-Relatively late (after pain).
-Ill defined.
- Rapidly growing.
- Warm & tender.

3-Fever & constitutional manifestations.

4-Pathological fractures: are rare.

Investigations:

1-Plain X ray:

a) Of the tumours shows:

- Expansion of the diaphysis with osteolytic lesion.
- New bone formation in the form of lamillated layers → *onion peel appearance.*

b) X ray chest: to detect pulmonary metastases.

2-Bone biopsy.

Differential Diagnosis:

From acute osteomyelitis: The toxaemia is mild, X ray shows the tumour & biopsy will ensure the diagnosis

Treatment: = combination therapy:

1- Preoperative radiotherapy & chemotherapy: as the tumour is very radiosensitive.
2- Amputation.
3- Postoperative chemotherapy.

Prognosis:

Prognosis is very bad The 5 year survival rate is about 5-15 %

6-Multiple Myeloma

Cells of origin:

Plasma cells of bone marrow.

Pathology:

Sites: -It is usually multiple from the start.
-It affects red marrow e.g. trunk, bones, skull, proximal humerus & femur.

MAC: Soft friable bone with multiple osteolytic lesions.

MIC: Sheets of plasma cells with characteristic large eccentric nuclei showing cartwheel appearance.

Complications:

1- Spread to involve liver, spleen, & LNs.

2- Myeloma kidney, & renal shutdown by protein cast.

3- Pathological fractures.

4- Hypercalcaemia → metastatic calcification.

Incidence:

It is common in middle aged 40-60 years.

Clinical picture:

General weakness, bony aches, & pathological fractures.

Investigations:

1-Plain X ray: multiple punched out bone defects (osteolytic lesions) with osteoporosis.

2-Urine analysis: *Bence Jone's proteinuria* that coagulates at 55°C and disappears at 85°C and reappears on cooling.

3-Bone marrow picture: though sternal puncture myeloma cells (plasma cells constitute 10% or more of the nucleated cells).

Differential diagnosis:

1- Bone secondaries.

2- Parathyroid osteodystrophy.

3- Osteoporosis.

Treatment:

- Radiotherapy & chemotherapy.
- Internal fixation of pathological fractures.

7- 8) Reticulum cell sarcoma & leukaemias

See general

Bone secondaries

Aetiology:

Malignant secondaries reach the bone either through:

1- Blood stream: (the commonest) from primary tumours elsewhere.

2- Direct spread: from nearby malignancy e.g. invasion of the mandible by tongue carcinoma.

Pathology:

Sites: - They are common in bones rich in red marrow.

1- Skull, sternum, ribs, pelvis, & vertebrae.

2- Diaphysis of long bone in case of severe anaemia as the yellow bone marrow of the shaft is replaced by red bone marrow.

Mac: there are three types:

1- Osteolytic secondaries: (the commonest).

2- Osteosclerotic secondaries as in cancer prostrate some cases of breast & adrenal carcinomas.

3- Diffuse infiltrating type: the malignant cells replace the bone allover the skeleton without new bone formation (cancerous osteomalacia).

MIC: As primary tumors.

Complications:

1- Pathological fracture.

2- Insomnia due to pain.

3- Severe anaemia: due to destruction of bone marrow.

Clinical picture:

1- Clinical picture of the primary tumour (missed in 10 % of cases).

2- Bony pain which is characteristically severe & persistent.

3- Rarely palpable bone swelling(s).

4- Picture of complications.

Investigations:

1- Plain X ray: It shows osteolytic or osteoscleotic single or multiple.

2- Bone scan: the most helpful (using ^{99m}Tc HDP Hydroxymethylene DiPhosphonate).

3-Investigations to detect the primary tumour if it is unknown.

Treatment:

I- Treatment of the primary tumour (inoperable).

II- Radiotherapy & chemotherapy.

III– Hormonal therapy: if the primary is hormone sensitive e.g. prostate & breast.

IV– Internal fixation of pathological fracture.

Bone cysts

1- Simple bone cyst:

- It is a unilocular cyst filled with clear fluid.

- It is more common in the metaphysis of long bones.
- It may cause pathological fracture.
- It tends to heal spontaneously, injection of steroid may help obliteration otherwise curettage and filling the cavity with bone chips.

2- Aneurysmal bone cyst:

- It is multilocular cyst filled with blood.
- It is more common in the long bone metaphysis but may occur elsewhere.
- It is best treated with curettage and filling the cavity with bone chips.

3- Cysts of hyperparathyroidism:

See later.

4- Hydatid cyst:

See before.

5- Fibrous dysplasia:

- It is a devlopmental disorder of bone characterized with replacment of bone medulla with fibrous tissue and possible cysts formation.
- It may predispose to deformity or pathological fracture.
- It may turn to fibrosarcoma in 5-10 % of polyosteotic fibrous dysplasia.
- Treatment is for complications. Localised lesion is curetted and filled with bone grafts.

Hand injury

Aetiology:

1- Industrial accidents (the commonest)

2- Home accident

3- Road traffic accident

Pathology:

Hand Injury may include on or more of the following

1- Skin injury (incised wound, crush wound, degloving injury & loss)

2-Tendon injury: partial or complete

3- Vascular injury (partial or complete cut, crushing)

4- Nerve injury (neuropraxia, axonotemesis, and neurotemesis)

5 Bone injury: fractures

6- Joint injury (ligaments, capsule &synovial membrane)

Complications:

The hand is a precious organ of functional mobility

1- The common dramatic complication is impaired movement in full range and/or power. Variable degrees of stiffness jeopardize function

2- Infection: if it occurs, it adds to tissue damage & stiffness

3- Gangrene: usually secondary to vascular impairment

4- Other complications of wound healing e.g. keloid, contracted scar & ulcers.

Incidence: -

The hand is the commonest part of the body injured in home &industrial accident. It is more common in manual workers

Clinical picture: -

- History of trauma
- Various types of injury usually with considerable bleeding and limitation of movement.
- Examine the hand for:

1) Extent of injury:

*skin, tendon, nerve, muscle, bones & joints.

*Vessels: ischaemia means urgent intervention

* Skin for viability & loss.

* Tendons & muscles injury is detected by loss of movement

* Nerve injury is detected by sensory, not by motor loss as the later may be due to tendon

injury, bone fracture or pain

- Bone injury (fracture) is detected by deformity, crepitus (risky) & best by radiological exam
- Joint injury is detected during exploration of the wound

(2) Viability of tissues (degloved skin, crushed muscles .... etc.)

(3) Presence of foreign bodies

(4) Degree of contamination

Investigations: -

1- Plain X-ray: to detect fractures & radio-opaque foreign bodies

2- MRI to detect the degree of soft tissue injuries.

3- Investigations to detect other injuries

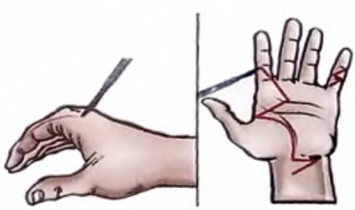
Treatment: -

ATLAS: (see before)

Repair = Definitive ttt

General rules for hand surgery

1) General anaesthesia is preferred but local IV anaesthesia may be used
2) Tourniquet is essential to get a bloodless field
3) Meticulous removal of foreign bodies and wound lavage is mandatory
4) Careful wound debridement & removal of non-viable tissue is important



Accepable incisions in the hand

5) Drainage with soft drains may be needed & mild compressive dressings are used to close the dead space but tight bandage should be avoided
6) Incisions in the hand should not cross perpendicular to skin creases. They are either zigzagged across lines of tension or run longitudinally in the neutral zones i.e. the lateral incision that joins the lateral limits of flexion & extension creases of the digits
7) The injured structures are repaired in the following order

I- Bone: fixation usually with Kirchner wire

II- extensor tendons

III- Dorsal venous arch

IV-Dorsal skin

Chapter VI

Hand & Foot surgery

V - Flexor tendons repair: flexor digitorum profundus is sufficient. Repair of flexor tendons in the area extending from the distal palmar crease to just distal to proximal interphalangeal joint is associated with high incidence of tendon adhesions and poor function so it is termed (No man's land)

No man's land

VI) Digital nerves: to bring the digital vessels into alignment

VII) Digital arteries

VIII) Volar skin: if there is skin loss, we can do

a- Partial thickness (Theirsh' s) skin graft

b- Full thickness (wolfe's) skin graft

c- Pedicle flaps e. g rotational flap, cross finger flap. etc

d- Microvascular free flaps

Cross-finger flap

8) Adequate haemostasis is essential

9) Splint & immediate elevation of paramount importance to avoid pain & oedema and favor healing

10) Early physiotherapy is mandatory to restore function & reasonable range of movement

N.B In finger injury special attention should be paid to the thumb being the most important finger (about 50% of the hand function) then to the index, middle finger, ring finger & little finger in that order

Hand infection

(Whitlows (داحوس))

It is one of the commonest infections in the body especially in manual workers.

Aetiology :

*Causative organisms: Staph aureus in 90% of cases.

*Route of infection: -Organisms usually get entry through punctures & minor wounds.

-Rarely, blood borne infection is the route.

*Predisposing factors : it is more common in:

-diabetics & immunocompromized patient.

-manual workers, housewives & dish washers.

Pathology

Types = Classifications:

I-Superficial infections:

1-Paronychia.
2-Subungual abscess.
3-Subcuticular abscess.
4-Pulp space infection (felon).
5-Web space infection.
6- Dorsal aspect infection.

II-Deep infections:

A-Spaces infections:

1-Thenar & hypothenar space infection.

2-Midpalmar (deep palmar) space infection.

3-Parona space infection.

B-Synovial sheaths infection:

1-Suppurative tenosynovitis of the fingers (intrathecal whitlows).

2-Ulnar bursitis.

3-Radial bursitis.

C-Bone & joint infections: - Osteomyelitis.

III-Unclassified infections

e.g. Infected wounds , & bites, pilonidal sinuses of barbars.

General rules in diagnosis & management of hand infection

1-Hand infection once suspected, antibiotic (penicillinase resistant) must be given.

2-No surgical intervention before adequate localization & exact location of pus.

3-*Don't wait for fluctuation* in palmar aspect infection as the thick skin & tough fascial septa delay fluctuation & increase tension (risky). The first sleepless night due to pain is an indication for immediate operation.

4-Oedema of the dorsum of the hand is marked even in palmar infection. *So, don't incise in the dorsum except after exclusion of palmar infection.*

5-Adequate anaesthesia is essential either general anaesthesia (better) or regional nerve block. Ring anaesthesia may be used in finger infection *but adrenaline must not be used to avoid ischaemia & gangrene of the finger.*

6-Tourniquet may be used to have a bloodless field.

7-Incisions should be done:

a)Over the maximum point of tenderness tested by a probe.

b)Never crossing the skin creases.

c)Respecting important structures e.g. vessels & nerves.

Position of immobilization

8-Drainage is secured by trimming the wound edge or by soft drain. Tough drains may press over vessels & nerves . Packs are contraindicated.

9-Immobilization & elevation are important to relieve pain & oedema & help healing. Immobilization is done in *Ideal position of immobilization* : the fingers are flexed 90° at metacarpophalangeal joints and minimally flexed at the interphalangeal joints. The thumb is adducted & the wrist is dorsiflexed. This position avoids or minimizes stiffness.

10-Early postoperative physiotherapy is essential.

I-Superficial infections

1-Paronychia.

Definition:

Infection under the nail fold.

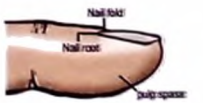
Aetiology:

1-Usually after removal of hang-nail or careless nail paring.

Clinical Picture:

-It is the commonest form of hand infection.

-Red tender swelling of the side of the nail fold which may extend to the other side " *run around* " & may extend under the nail → subonychia.



Anatomy of the nail

Paronychia & run around

Treatment:

-Drainage of pus by Hilton's method.

-If a pocket is present under the corner of the nail fold a triangular area of the nail fold is excised.

-In subungual abscess, part or the whole nail is excised.

Drainage of paronychia

Chronic paronychia : is a more insidious infection of the nail fold in house wives & dishwashers . Fungi e.g monilia are the commonest organism . So antifungal agents both orally & locally give good result beside keeping the fingers as dry as possible.

2-Subungual abscess
(Subonychia)

Aetiology:

1-Pricks beneath the nail (apical). 2-Extension from paronychia (basal).

Clinical picture:

-It appears as white area under the nail.

Apical abscess

Treatment:

-V shaped incision for apical abscess to evacuate pus & remove FB if present

-Basal abscess and floating nail usually need nail extraction.

3-Pulp space infection.

(Felon) (داحوس في الإصبع)

Anatomy of pulp space:-

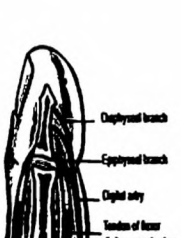
-It is a closed space bounded by:

*skin in its palmar, lateral & distal aspects.

*distal phalanx posteriorly.

*proximally the distal palmar crease fuses with the deep fascia connecting it to the base of distal phalanx.

-It contains fat that is traversed by fibrous septa that divide it into about 16 compartments.



Anatomy of pulp space

-The space doesn't contain tendons or synovial sheathes.

-The digital artery gives an epiphyseal branch to the epiphysis of the distal phalanx before it enters the pulp space. So, if it develops septic thrombosis, 2ry to pulp space infection only the diaphyseal branches are affected while the epiphysis escapes necrosis and may regenerate a new diaphysis.

Aetiology:

1-Usually pin pricks. 2-Rarely extension from paronychia.

Clinical picture:

-It is the second commonest form of hand infection.

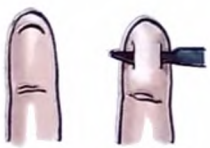
-Pain, early is dull aching, but later, it becomes severe & throbbing.

-Swelling, , hotness, minimal redness & severe tenderness.

-Fluctuation is late & may occur only after necrosis.

Treatment:

Don't wait for fluctuation



Old (wrong) incisions

1-Old incision:

a-Fish mouth & transverse incision.

b-Lateral (hockey stick) incision that is deepened to cut all fibrous septa & open all compartments. A counter-incision may be needed.

These incisions have rendered too many finger tips gangrenous or anesthetic.

Recent (correct) incisions

2-Recently:

By a small longitudinal or oblique incision that is done over the point of maximum tenderness, but not across the flexion crease. It respects the neurovascular bundles.

4-Subcuticular abscess

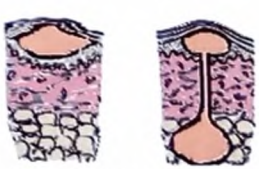
Aetiology:

1-Usually due to pinprick (infected blister).

2-pointing subcutaneous abscess → *Collar stud abscess*.

Clinical picture:

It appears as yellow infected blister.



Subcticular & collar stud abscess

Treatment :

-Deroofing of the superficial collection and examine the floor for a tract. If present, it is followed to drain the deep abscess.

5-Web space infection

Definition:

Infection of the superficial spaces inbetween the bases of the fingers.

Web space infection

Aetiology:

1-Due to pin pricks.

2-Rarely, extension from deep palmar space infection through lumbrical canals.

Clinical Picture :

-Painful, red, hot, tender, swollen space causing separation of fingers.

Treatment:

-It is drained through transverse palmar incision over the maximum tender point.

-Counter-incision may be needed on the dorsal surface.

Incision

6-Dorsal aspect infection

It resembles infection anywhere in the body, but oedema is marked as the tissues are lax.

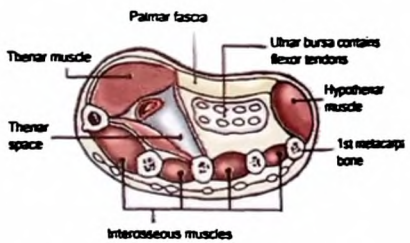
II- Deep infections

A- Volar spaces infection.

1-Thenar space infection.

Thenar space is bounded:

- Anteriorly by palmar fascia.
- Posteriorly by adductor polices muscle.
- Laterally by the thenar muscle.
- Medially by a fibrous septum connecting the palmar fascia to the third metacarpal.



Anatomy of the palmar spaces

Aetiology:

1-Through penetrating trauma.

2-Extension from tenosynovitis of radial bursa.

Clinical picture:

-Painful, red, hot, tender swollen space with marked oedema of the dorsum.

-The cup of the hand is preserved.

Treatment

1-Old incision along the radial border of the 2nd metacarpal bone on the dorsal aspect but this incision may injure the lateral branch of median nerve.

2-Recent incision: by a curved or zigzag incision on the web between the thumb & index fingers & the collection is entered by the tip of forceps & drained by Hilton's method.

Incisions for thenar space

2-Deep midpalmar space infection.

This space is bounded:

-*Anteriorly* by the ulnar bursa containing the flexors tendons of the medial three fingers.

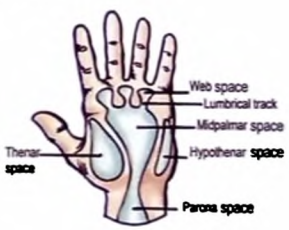
-*Posteriorly* : by the third, fourth, fifth metarcarpal bones and the interossei muscles inbetween.

-*Laterally* : by the fibrous septum of the thenar space

-*Medially* by the hypothenar space.

-*Proximally*, it communicates with space of Parona.

-*Distally*, it communicates with the web spaces through the lumbrical canals.



Volar spaces infection

Aetiology:

1-direct through a pinprick or stab.

2-extension from web space or ulnar bursa infection.

Clinical picture:

-It is a rare condition (1% of all forms of hand infection).

-Painful, red, hot, tender swelling that obliterate the cup of the hand.

-Oedema is marked on the dorsum of the hand.

Drainage of midpalmar space

Treatment:

The midpalmar space is better drained through incision in the 3rd or the 4th web then the space is entered through the lumbrical canals by Hilton's method.

N.B, Incisions in the web shouldn't exceed the distal palmar crease so as not to open ulnar bursa or injure superior palmar arch.

3-Parona space infection

(Forearm space)

Anatomy:

Parona space lies in the distal forearm between pronator quadratus muscle dorsally & flexor tendons ventrally.

Drainage of Parona space infection

Aetiology:

Due to spread from palmar space infection or from ulnar or radial bursitis.

Clinical picture:

It usually presents as extension of hotness, redness & swelling to the forearm from the original hand infection.

Treatment :

Drainage through longitudinal incision on the radial or ulnar border of the space. If infection spreads up in the forearm, another ulnar incision in the middle 1/3 of the forearm .

B-Tendon sheath infection

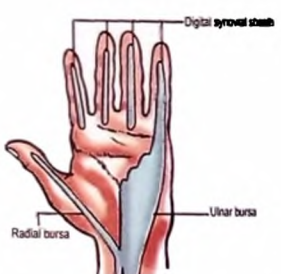
(Tenosynovitis)

Anatomy of synovial sheaths:

-The flexor tendons sheaths of the middle three fingers extend only to the metacarpophalangeal joints. While the flexor tendon sheath of the little finger is continuous with the ulnar bursa

-Ulnar bursa is the synovial sheath that surrounds the flexor tendons of the medial four fingers from the distal palmar crease to about one inch above the wrist joint.

-The flexor tendon of the thumb continues upward as the radial bursa that communicates with the ulnar bursa in about 50% of population.



Anatomy of synovial sheathes

Aetiology:

1-Direct through pin pricks .

2-spread from infected palmar spaces.

Clinical Picture

1-Tenosynovitis of the middle three fingers: There are

a) Throbbing pain over the inflamed sheath.

b) Fusiform swelling of the affected finger.

c) Moderate flexion of the finger with severe pain on passive extension.

d) There is sharp localized tenderness over the affected sheath (pencil point tenderness).

Kanavel's point

2-Ulnar bursitis: There are:

-Throbbing pain, hotness, redness & swelling over the sheath.

-It is differentiated from deep palmar space infection by:-

a) The cup of the hand is not obliterated.

b) Swelling & tenderness involve the little finger with maximum tenderness over the part lying between the two transverse palmar creases *(Kanavel's sign)* .

Drainage of tenosynovitis

3-Radial bursitis : There are

throbbing pain, hotness, redness & swelling over the course of radial bursa that is differentiated from thenar space infection by

a) The pencil point tenderness.

b) Involvement of the thumb.

Treatment:

-They are best drained through transverse incisions over their ends and intra-operative irrigation of the sheaths with saline, antibiotics and local anaesthetic through small (1mm) ureteric catheter threaded into them. They should be thoroughly irrigated.

- Ulnar bursa may be also explored through Henry's incision along the ulnar border of the hand.
- Radial bursa may be explored through curved incision along the medial side of the thenar eminnce that should stop at least half an inch distal to the flexor retinaculum not to injure the lateral branch of the median nerve supplying the thenar muscles.

C-Bone & Joint infection

- Osteomyelitis is rare. It commonly affects the phalanges especially the distal due to neglected pulp space infection.
- Septic arthritis may complicate open wounds or suppurative tenosynovitis. Stiffness follows in about 50% of cases.

Foot infections

Aetiology

1-The organisms usually get entery through superficial cracks or wounds

2- It may be secondary to ingrowing toe nail or complicate ischaemia

Pathology

Types = classification

I- Superficial Infection

1-paronychia	2-subungual abscess	3-subcuticular abscess (common)
4-pulp space	5-web space	6-Heel space infection

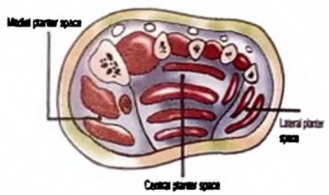
II Deep infection

A- Plantar space infection

1- Medial plantar space infection

2- Lateral plantar space infection

3- Central plantar spaces infection there are four central spaces arranged among the four layers of the short muscles of the foot



Plantar spaces infection

B-Synovial sheaths infection

1-Digital sheaths	2-Sheath of peroneus longus	3-Long flexors sheaths
5-Tibialis anterior sheath	4-Long extensors sheaths	6- Tibialis posterior sheath

C-Bone & joint infection

Osteomyelitis & septic arthritis are common in diabetic patients

Clinical Picture:

As in hand infection

Treatment:

- It follows the same general rules in management of hand infection but the functional disability that may complicate infection or surgery is much less common i.e one or more tendons may be sacrificed without much functional impairment

-Drainage is usually through longitudinal incisions in the plantar aspect

Drainage of plantar infection

Ingrowing toenail ظفر غائر

Aetiology

Due to encasing sweaty feet in tight shoes

Predisposing factors

-Familial tendency

-sweaty feet, thin nail plate, fleshy nail folds

Ingrowing nail

Clinical Picture

Painful tender, swollen, hot, red nail fold – paronychia

Wedge resection

Treatment

Usually surgical by wedge resection of the affected nail fold and part of the nail including the bed with caustic application to the bed to prevent recurrence

Diabetic foot infection & gangrene

There are two major types of diabetic gangrene:

I- Diabetic atherosclerotic (ischaemic) gangrene

(Diabetic macroangiopathy)

- Diabetic patients are more prone to atherosclerotic changes at earlier age group.
- Thrombosis & obstruction affect major vessels of the limb.
- There is no distal pulsation.
- The gangrene begins dry but commonly becomes infected.

Clinical picture , Investigations , & treatment as in chronic ischaemia.

N.B. Sympathectomy is useless in diabetics as diabetic neuropathy acts as auto-sympathectomy.

II- Diabetic infective gangrene

(Diabetic microangiopathy)

Pathology:

Site: the commonest site is the foot because:

1- It is a common site for trauma which is usually neglected.

2- It is a common site for ischaemia & peripheral neuropathy.

3- It is a common site for infection (tinea pedis).

The tissues are very vulnerable to infection as diabetes causes:

1-Diabetic microangiopathy: affecting distal arterioles & capillaries causing tissue ischaemia.

2- Diabetic neuropathy: Diabetic peripheral neuritis favours infections due to:

- Negligence of minor trauma.
- Thick callosities & trophic ulceration.

3- Compromised immunity: both humoral & cell mediated immunity are disturbed. Also, the tissue fluids are saturated with glucose which is a good media for infection.

Incidence:

It is common in middle age or elderly females with uncontrolled diabetes.

Clinical picture:

- History: of uncontrolled diabetes & history of minor trauma.
- Signs of foot infection and /or gangrene.
- Signs of inflammatory reaction.
- Palpable nearby pulsation (dorsalis pedis & posterior tibial arteries).

Treatment:

I- Conservative treatment:

- Proper control of diabetes using insulin.
- Broad spectrum antibiotics.
- Drainage of all pockets of pus and adequate debridment of all sloughed tissues.
- Repeated dressing with glycerin magnesia till granulation & healing.

II- Amputation:

Indications:

1-established gangrene. 2- Spreading infection. 3- osteomyelitis.

- Conservative amputation for dry gangrene and urgent amputation for spreading gangrene.
- The stump may be left open for 5 days to drain infected tissue fluid then closed.

Chronic leg ulcers

1-Chronic traumatic ulcer,

Aetiology: wounds. Burns, radiation ulcer, bedsores

Pathology: *Single or multiple* *Sensation:* Painless unless infected

Site: commonly over bony surfaces e.g. over chin of tibia

Size: variable *Shape*: rounded, oval or irrigular

Floor: healthy or unhealthy G.T *Base*: indurated

Edge: punched out *Margin* : pigmented .

Discharge: serous or seropurulent *Draining LNs* enlarged firm & tender

2-TB ulcer: multiple , painful, undermined with soft base, cyanotic margin and pale granulation tissue floor. Mostly over deep T.B. lesion .

3- Syphilitic ulcer: usually single, punched out, painless commonly over the middle 2/4 of the leg.

4-Chronic osteomyelitis ulcer .

It is the mouth of osteomyelitis sinus (see orthopedic) .

Venous ulcer

5-Venous ulcer:

(See before).

6- Arterial ulcer (ischaemic ulcer)

Aetiology: chronic ischaemia e.g. atherosclerosis & Burger's disease.

-It commonly occurs in toes and feet with evidence of ch. ischaemia.

-It is resistant to treatment. Sympathectomy may be beneficial.

Arterial ulcer

7- Lymphoedema ulcer:

Aetiology: Due to rupture of lymphoedema vesicles (see before).

8- Neurotrophic ulcer:.

Aetiology: peripheral nerve injuries, peripheral neuritis, cord lesions, leprosy ...etc

Pathology: It starts as callosities over the pressure areas e.g. heel or the ball of big toe which is neglected. An adventitious bursa develops under it which becomes infected and abscess is formed .rupture or drainage of the abcess leaves a deep ulcer penetrating down to bone. The ulcer is painless and surrounded with an area of anaesthesia .

Neurotrophic ulcer

9- Ulcers complicating haemolytic conditions:

as haemolytic anaemia , e.g. cong. Spherocytosis , thalassaemia .

10- Malignant ulcer:

1- Primary skin cancer e.g. squamous cell carcinoma, melanoma. etc

2- Ulcerating deep malignancy e.g. fibrosarcoma, osteosarcoma .. etc

Chapter VII

Head & Neck Surgery

The Scalp, Skull & Brain

The scalp

Anatomy

-The scalp is the soft tissues that cover the skull from the eye brow to the superior nuchal line and from one zygomatic arch to the other.

-The scalp is formed of the following layers:

1-Skin: which is very rich in hair follicles & sebaceous glands.

2-Connective tissue: It represents the subcutaneous connective tissue, which is dense.

3-Aponeurosis: epicranial aponeurosis or galea aponeurotica which gives an insertion for both the frontal & occipital bellies of fronto-occiptalis muscle.

4-Loose connective tissue: Areolar connective tissue loosely connects the galea to the periosteum. Through this layer the previous layers can be separated or avulsed from skull.

5-Pericranium: which is the periosteum of skull bones that dips in the suture line to form what is called suture membrane that blends with the periosteum of the inner table of the skull.

Traumatic conditions

I-Closed wounds

1-Scalp haematoma

Aetiology:

It is due to blunt trauma to the head.

Pathology:

Sites or types:

1-Subcutaneous haematoma:

-It is localized to the site of trauma and forms a small painful swelling.

-It may have a depressed center like depressed fracture of the skull but it moves with the scalp over the skull.

2-Subgaleal (Subaponenrotic) haematoma:

-It forms diffuse haematoma under the galea aponeurotica.

-It extends as far as the attachment of the galea:

a) anteriorly to the supraorbital ridges.

b) posteriorly to the superior nuchal lines.

c) laterally to the zygomatic arches on both sides.

-It forms a soft fluctuant swelling over which the scalp floats.

3-Subperiosteal haematoma or Cephalohaematoma:

-Haematoma below the periosteum (pericranium) of the skull bone.

-It extends only to the suture lines, as the periosteum is attached to the sutural membrane so, it takes the shape of the underlying bone.

-It has a soft cystic consistency but margin may be raised and indurated due to fibrin deposition & clotting at the periphery .The center remains soft & depressed simulating a depressed fracture of the skull but, the edge is indentible in haematoma & hard in depressed fracture.

-It is commonly caused by head injury during birth but may collect from an extradural haematoma in the temporal region through the fracture forming a safety valve haematoma that may delay manifestations of compression.

Treatment:

-They usually respond to the conservative measures in the form of cold fomentation in the first 24 hours & hot fomentation in next days together with antibiotics & analgesics.

-Subgaleal haematoma usually needs aspiration and bandage.

2-Ecchymosis.

It is a milder form and responds to conservative treatment.

II-open wounds

Pathological types:

1-Incised wounds are caused by sharp instruments e.g. knife or axe.

2-Lacerated (contused) wound: (the commonest) due to blunt trauma to the scalp. As the skin is tough & stretched over the skull, the edges of the wound may be clean cut simulating an incised wound.

3-Firearm wound: The scalp wound is lacerated with fracture of the skull with the characteristic loss of substance and internal & external beveling.

-In open wounds of the scalp, the bleeding is profuse because the scalp is rich in blood vessels and the dense subcutaneous tissues anchors the blood vessels and prevents them from retraction & contraction holding them open.

Management: (see mangement of polytraumatized patient in chapter I)

I- **Primary survey:** to detect what is killing the patient.

II- **Resuscitation (A, B, C, D, E):** to manage what is killing the patient

*Arrest of bleeding is achieved temporary by applying multiple Allis forceps to the bleeding edge and everting it outwards to kink the blood vessels and stop the bleeding.

III- Secondary survey: to detect other possible injuries.

*Examine for possible fracture of the skull or intracranial injury (Signs of lateralization).

IV- Definitive management:

-Clean the wound and remove any foreign bodies & shave the hair around.

-Irrigate the wound with sterile saline.

-Trimming of the wound edge is advisable in contused wounds.

-Suturing the wound with single layer of silk sutures through all layers.

-In case of tissue loss, undermining of the edges or using local flaps may enable closing the defect without tension.

-Avoid rubber drains for the fear of infection and if mandatory, use suction drains and crépé bandage.

Neoplasms

I-Benign: Lipoma, papilloma, haemangioma, cirsoid aneurysm, plexiform neurofibroma, dermatofibroma, keratoacanthoma.

II-Locally malignant: Basal cell carcinoma e.g. Turban tumour.

III-Malignant:

Primary: Epithelioma, melanoma, sebaceous adenocarcinoma & dermatofibrosarcoma.

Secondaries: e.g. secondaries from follicular carcinoma of the thyroid (pulsating secondaries).

The Skull

Congenital Anomalies

1- Cephalocele(Cranium bifidum).

Definition:

It is herniation of the dura with or without brain tissue through a congenital defect in the skull.

Pathology:

1-Meningocele:

Site: common in the occipital region.

Composition : It is formed of the dura and contains CSF.

Meningocele

2-Encephalocele:

Site: common in the root of the nose.

Composition: It is formed of herniated brain tissue.

-The herniated brain tissue may contain part of dilated ventricle and called *hydroencephalocele.*

Encephalocele:

3-Meningo-encephalocele :

Site: common in the occipital region.

Composition: It contains brain tissue surrounded by CSF.

Complications:

1- Rupture with fatal meningitis.

2- Hydrocephalus is a common associated anomaly that usually manifests after surgical excision.

Clinical picture: -

1-Meningocele:

- It is soft cystic translucent swelling.
- It is compressible on pressure but this may induce vomiting as it increases ICP suddenly.
- It is cross-fluctuant with the anterior fontanelle.
- It gives impulse on cough or crying and becomes larger and more tense.
- It pulsates with respiration (not with heart beats).

2-Encephalocele:

It differs from meningocele:

- It is less soft in consistency and opaque in transillumination.

-It is associated with marked neurological manifestations e.g. spastic paralysis.

3-Meningo–encephalocele:

It resembles encephalocele with high incidence of associated hydrocephalus.

Treatment:

Surgical excision & closure of the defect. To avoid hydrocephalus that always develops after the operation, ventriculo-atrial or better ventriculo-peritoneal shunt is carried out prophylactically.

2- Hydrocephalus

Definition:

It is dilatation of the ventricular system of brain due to accumulation of excessive amount of CSF.

Normal CSF circulation

CSF is secreted by the choroid plexus in the lateral ventricles of the brain. CSF passes from there through *foramen of Monro* on each side to reach the third ventricle in the midline then through *the aqueduct of Sylvius* to the fourth ventricle between the brain stem and cerebellum. CSF escapes from there through two *lateral foramina of Luscka* and one midline *foromen of Magendi* to the subarchnoid space (SAS) around the brain and spinal cord. CSF is absorbed mainly by the arachnoid villi into the blood stream.

Aetiology:

I– Congenital:

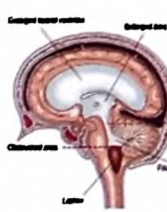
1) Congenital stenosis or forking of aqueduct of Sylvius.
2) *Arnold Chiri malformation*: caudal dispacement of the brain stem into foramen magnum obstructing the SAS. This is commonly associated with spina bifida and meningomyelocele.
3) Cerebral malformation as encephalocele.

II – Inflammatory: -

1) Toxoplasmosis causes stenosis of the aqueduct of Sylvius.
2) Meningitis and encephalitis.

III- Neoplasms:

1) Neoplasms of the fourth ventricle e.g. medulloblastoma,
2) Choroid plexus papilloma and tumours of the ventricles.



Non-communicating hydrocephalus

Pathology:

Types: The disease is commonly classified into:

1- Non-communicating hydrocephalus:

There is obstruction of the ventricular system either at the aqueduct of Sylvius or foramina in the fourth ventricle.

2- Communicating hydrocephalus: It is either due to:

a. Increased secretion of CSF (rare) e.g inflammation.
b. Block in the absorption of CSF at the arachnoid.

Communicating hydrocephalus

Complications:

1-Pressure atrophy of the brain with mental retardation.

2-Progressive wasting & increased susceptibility to infection.

3-Enlargement of the skull with separation of sutures.

4-Increased intra-cranial tension.

Sun set appearance

Incidence:

As an isolated congenital anomaly .It occurs in about 1/1000 live-birth and with other associated anomalies in about 1/1000 live birth.

Clinical picture:

I – In infants and children before closure of skull sutures:

The patient presents with:

1- Macrocephaly with visible dilated scalp veins.

2- tense non-pulsating fontanelle .

3- the eye globes are pushed downoward giving what is called *sun set appearance* with failure of upward movement (*Staring Look*) .

II- After closure of sutures:

The patient presents with manifestations of increased intra cranial tension.

1- Headache may awake the patient from sleep .

2- Vomitng which is projectile, not related to meals, and more at the morning.

3- Blurred vision and may be squint. Papilloedema appears on fundus examination.

Investigations:

1- Skull X ray: In infant, it may demonestrate separation of sutures (*silver beaten appearance*).

2- Injection of air or inert dye into the subarachnoid space of the spinal cord . If the air or the dye are recovered in the ventricles, hydrocephalus is considered communiceting and vice versa.

3- CT scan and MRI are the ideal investigations to diagnose hydrocephalus and detect its cause.

Treatment:

I- Conservative treatment: Almost all cases need surgery except:

1- Neglected cases with cerebral corrtex thickess less than 2 cm because the patient will be handicapped even after successful surgery.

2- Cases show spontaneous improvement (arrested hydrocephalus).

Medical treatment includes:

1) Acetazolamide is carbonic anhydrase inhibitors that reduces CSF production by about 60% .

2) Frusemide also has an effect on CSF production.

II- Surgical treatment

Shunt operations:

1- Ventriculo- atrial shunt: Inserting an inert valved catheter into the lateral ventricle through small burr hole and tunneling it under skin to reach the right atrium through SVC.

2- Ventriculo peritoneal shunt: as before ,but the distal end is inserted into peritoneum.

3- Lumbo peritoneal shunt: The catheter is inserted into the lumbar theca and the distal end is placed in the peritoneum.

Complications of shunt operation:

1) Infection.

2) Obstruction of the shunt.

Prognosis:

The prognosis depends on many factors such as:

1-general condition of the patient before operation.

2-the viable mass of the cerebral cortex remaining .

3-the circumference of the skull. The prognosis is poor if circumfaremec is over 19 inches.

Trauma

Fractures of the skull

I-Vault Fracture

Aetiology: Direct Trauma.

I- Blunt trauma:

1-Direct local trauma: direct blow. The fracture begins at the site of contact.

2-Compression trauma: compression of the skull by a hard flat surface may make the skull to burst at the maximum convexity.

II- Penetrating trauma: as bullet & missiles.

Pathology:

I- Fissure fracture:

- It is a linear fracture (crack) of the skull with no displacement due to blunt trauma.

II- Depressed fracture:

- The fractured bone is depressed below the vault. It may be:

1- Pond fracture: -

- It occurs in newly born and simulates green stick fracture in long bone.

-The contour of the vault is smoothly curved inward without displacement in the fractured ends.

2- Steep fracture:

When the fractured ends are depressed inward.

3- Comminuted fracture:

The bone is broken into several pieces, which are driven inward.

Complications: -

1- Compound fracture: It may lead to:

a) Leakage of CSF to outside if the meninges are also injured.

b) Osteomyelitis of the skull.

c) Intracranial infection e.g. meningitis.

Comminuted depressed fracture

2- Intracranial haemorrhage:

Extradural , subdural & intracerebral haemorrhage.

3-Brain injury:

Concussion, compression, contusion or laceration.

Clinical picture:

- History of trauma to the head.
- It is usually associated with scalp wound (compound fracture).
- The fracture may be felt or even seen through the wound.
- Signs of complications if present e.g. CSF leak & extradural haemorrhage.

Investigations:

1-Plain X ray: It appears in X ray as a radiotranslucent line that should be differentiated from:

a) Suture line which is known by:

- its anatomical position. - its zigzag course.

b) Groove for meningeal blood vessel which is known by:

- its anatomical position. - its arborizing course.

2- CT scan: To diagnose intracranial haemorrhage or injury.

Treatment:

I- Fissure fracture:

- Simple uncomplicated fissure fracture is left alone.
- Compound fracture is explored through the wound and , if there is no intracranial injury, close the wound as usual. If there is intracranial complications, deal with (see later).

II Depressed fracture:

1- Conservative ttt:

It is indicated in simple pond fracture as it usually corrects itself spontaneously within 6 weeks.

II- Surgical ttt:- indicated in:

1- Compound fracture.

2- If the depression is more than 3 mm in depth.

3- If the depression lies over speech or motor area.

4- If there is associated intracranial injury.

5- Pond fracture of the forehead in infant that fails to correct itself in 6 weeks.

Procedure:

1-Trephine is done beside the fracture and elevate the depressed fragments with elevator.

2- Loose foreign bodies & damaged brain tissue are removed.

3-Dura is examined (normally it is white, lustrous & pulsating). If the dura is torn, suture it. If the dura appears dark, dull with no pulsation, this means subdural haematoma, so evacuate it.

II-Fracture of the base

Aetiology:

I- Indirect trauma (more common):

1- Trauma to the vault: It causes either fissure fracture of the vault that radiates to the base or even the vault may escape injury because it has some elasticity while the base is fractured being rigid & weakened by multiple foramina

2- Trauma to the spine: As falling from a height. The spine may fracture the base of the skull at the posterior fossa.

3- Trauma to the mandible: The mandibular condyle may fracture the middle cranial fossa.

4- Trauma to the face: The maxilla may transmit trauma to the anterior cranial fossa.

II- Direct trauma: Through penetrating trauma to the mouth, orbit, nose & vault.

Pathology:

Site: It may be in the anterior, middle ,or posterior cranial fossa.

Types: It is usually compound fracture due to laceration of the mucous membrane of the nose, pharynx or ear communicating the fracture to outside.

Complications:

1- Compound fracture: leading to:

1) Escape of intracranial contents

a) CSF. b) blood. c) brain matter (rare).

2) Introduction of infection leading to meningitis.

2- Injury to cranial nerves: by three mechanisms:

i. Laceration at the time of trauma → immediate paralysis with no recovery.

ii. Compression by haematoma or exudate → paralysis after hours & recovery after days.

iii. Constriction by callus or scar tissue → paralysis after weeks & no recovery.

3- Brain Injury: Concussion, contusion, compression & laceration.

Clinical picture: according to the site of the fracture:

I- Anterior cranial fossa fracture

Raccoon's eyes

A- Escape of intracranial contents:

1) Blood: in the form of:

a) Epistaxis if the cribriform plate of ethmoid is involved.

b) Bleeding into the orbit to appear around the eye giving *Panda bear or Raccoon's sign* and gives a subconjunctival haematoma.

2) CSF: in the form of CSF rhinorrhea that gives continuous leakage through the nose with continuous salty taste due to high Cl^- content of CSF.

B- Picture of cranial nerves injury:

The 1st, 2nd, 3rd, 4th, ophthalmic division of the 5th & the 6th cranial nerves may be injured.

C- Picture of associated brain injury.

II- Middle cranial fossa fracture:

Battle's sign

A- Escape of intracranial contents:

1- Blood: in the form of :

i. Epistaxis if the sphenoidal nasal sinuses is involved.

ii. Bleeding through the ear.

iii. *haematoma around mastoid bone (battle sign).*

2- CSF, in the form of rhinorrhea & otorrhea.

B- Picture of cranial nerves injury:

The 5th, 6th, 7th & 8th cranial nerves may be injured.

C- Picture of associated brain injury.

III- Posterior cranial fossa fracture

Sites of CSF leak

A- Escape of intracranial content:

1- Blood: in the form of

i. Bleeding through the mouth (rare).

ii. Occipital haematoma in the back of the neck.

2- CSF: Through the pharynx (rare).

B- Picture of cranial nerves injury:

The 9th, 10th, 11th cranial nerves may be injured. The 12th cranial nerve is protected by the

strong condylar foramina & condyles.

C- Picture of associated brain injury.

Investigations:

1-Plain X ray: it rarely shows the fracture. Intracranial air may be present.

2-CT scan: it shows the fracture and detect associated brain injury.

Differential Diagnosis:

I- Traumatic rhinorrhea from running nose due to common cold.

Rhinorrhea due to CSF leak doesn't harden hand kerchiefs.

II- Orbital bleeding from direct trauma to the eye (black eye).

In orbital bleeding due to extension from anterior cranial fossa there is:

1-The bleeding & discoloration are delayed for hours or days after trauma.

2-The subconjunctival hage. is dark red, triangular in shape with the apex towards the cornea with non-visible posterior limit. Subconjunctival hage. due to local trauma is immediate, bright red, triangular with the base towards the cornea and has definite posterior limit.

III- Bleeding from the ear in fracture middle cranial fossa from bleeding due to rupture drum: In rupture drum, the blood clots but not in the former due to mixing with CSF.

Treatment:

I- Prevention of intracranial infection:

1- Rest in bed for 2-6 weeks with repeated observation & neurological assessment.

2-Massive doses of broad spectrum antibiotics given for 1week after cessation of CSF leak.

3-For rhinorrhea & otorrhea:

a) Plug the ear or nose with clean sterile cotton & warn the patient from blowing his nose.

b) Nasal or ear washes are contraindicated.

c) If leak persists for more than 10 days, surgery is indicated to repair the torn dura may be with fascial graft e.g. fascia lata.

II- Treatment of cranial nerve injury:

-Treatment is usually conservative.

-Surgery is indicated in delayed injury due to compression on the nerve by callus or scar tissue e.g. facial nerve decompression in the facial canal.

III- Treatment of associated brain oedema: see later.

Prognosis:

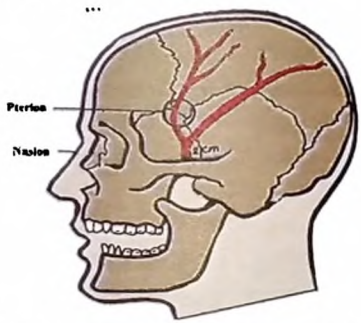
The mortality rate is 25% in the anterior fossa, 50% in the middle fossa & 75% in the posterior fossa fractures.

Intracranial haemorrhage

1- Extradural haemorrhage

Anatomy of middle meningeal artery

- It arises from the maxillary artery in the infratemporal fossa.
- It enters the cranial cavity though the foramen spinosum.
- It divides into two branches:
  - The anterior branch that passes on its bony canal in the inner aspect of the temporal bone.
  - The posterior branch passes upward and backward to the occipital bone.



Surface anatomy of the middle meningeal artery

Aetiology:

I- Causative trauma:

Direct trauma to the head especially in the temporal region. In adults there is usually vault fracture but may be absent in children.

II – Sources of bleeding:

1- Diploic veins: it is the commonest source.

2- Venous sinuses of the dura.

3- The middle meningeal vessels (the most serious):

The anterior branch of the artery is more commonly injured than the posterior one due to:

1) The artery runs in a bony canal easy to be injured during fracture.

2) The squamous part of the temporal bone is thin & easy to be fractured.

Pathology: There are three stages:

1) Stage of concussion:

- It is due to sudden loss of brain function.
- The injured vessel may not bleed or bleeds minimally due to hypotension.

2) Lucid interval:

- The patient recovers from concussion.
- The bleeding contiues and blood accumulates in the extradural space.

3) Stage of compression:

The accumulated haematoma compresses the brain.

Clinical Picture:

I- Stage of concussion :

- Unconsciousness. - slow weak pulse. - hypotension.
- Slow shallow respiration. -severe pallor. -dilated pupil.

II – Stage of lucid interval:

- The patient regains consciousness and even may continue his daily activities.
- It varies in length from minutes to several hours.

III -Stage of compression:

A- Early: There is irritation of the brain leading to:

- *Headache, irritability & drowsiness.*
- *Vital signs:* *Pulse: slow full. *BP: hypertension.
  *Temp: subnormal temperature. *RR: slow deep.
- *Localizing signs:* Convulsion of the contralateral side of the body.

B- Late: there is paralysis & loss of function leading to:

- *Coma.*
- *Vital signs :* *Pulse : rapid & weak. *Bl. pr. : hypotension.
  *Temp.: fever. *RR : Chyne Stoke's respiration.
- *Localizing signs:* Hemiplegia of the contralateral side.

The pupilary changes:

On the same side	On the other side
Miosis	normal
Mydriasis	normal
Mydriasis	Mydriasis

- Lastly there is decrebrate rigidity & death.

Investigations:

1- Plain X-ray: to detect skull fracture (vault).

2- C T scan & MRI: are very helpful.

Treatment:

I- Management of head injury. (mention)

II- Urgent operation:

- Vertical incision in the scalp in the temporal region begning at the midpoint of the zygomatic arch.
- Temporalis muscle is splited and the periosteum of the temporal bone is elevated.

Trephine holes

- Burr holes are made:

1) over the pterion (2 inches above the middle of the zygomatic arch) for anterior branch.

2) At a point 2 inches above the tip of mastoid process for the posterior branch.

-Trephine holes are enlarged with bone nippling forceps .

- Deal with the bleeding vessels.

1)Diploic veins: crushing the two tables of skull over them or better than bone wax tamponad.

2)Venous sinuses: suturing the tear in the wall better over muscle graft (from temporalis) that contains kinase & thrombin that help clotting & haemostasis.

3) Middle meningeal artery: -bone wax at foramen spinosum.

- underruning sutures or silver clips.

- Examine the dura for subdural haematoma. If dark, dull & non-pulsating, open it and evacuate haematoma with a sucker, secure haemostasis & close it.

Signs of lateralization

The site of extradural haematoma (compression) is usually:

1- The side of haematoma in the temporal region .

2- the side of fracture of the skull.

3- the side of the 1st pupil to dilate.

4- The contralateral side of hemipelgia and convulsion.

2- Subdural haemorrhage

Aetiology:

3- Rupture of cerebral veins where they traverse the subdural space.

4- Rupture of the dural venous sinuses.

A- Acute subdural haemorrhage:

- It forms and accumulates rapidly at the time of trauma.
- It is usually diffuse compressing both halves of the cerbral cotex.
- It is commonly associated with brain contusion, laceration and marked oedema.
- Clinically, there is no lucid interval and the the patient passes directly from stage of brain concussion into brain compresssion and the level of consciousness deteriorates progressively.
- Surgical evacuation is done through burr holes. Open the dura and wash the subdural space.
- The prognosis is worse than extradural haemorrhage due to associated brain injuries. The mortality rate is about 50%.

B- Chronic subdural haematoma:

- It forms slowly after mild head trauma usually in old persons.
- It will undergo haemolysis that increases its osmotic pressure so, it imbibes fluids from the surroundigs and increases in size. Accordingly, compressing of the brain ensue.
- Clinically, the symptoms appear a long time after trauma that may be forgotten by the patient, usually in the form of headache, dizzness and hemiparesis.
- CT scan is diagnositic.
- Surgical evacuation is usually needed.

2- Subarachnoid haemorrhage

- Mild hage occurs in most cases of head trauma and explains post-concussion headache.
- Massive hage may orginate from the internal caroted or its branches and is rapidly fatal.

3- Intracerebral haemorrage

- They commonly occur with brain laceration. The prognosis depends on the size of haematoma and the site & the degree of brain damage
- Rupture of intracerebral artery leading to intrcerebral haematoma with minimal brain damage.

Management of head injury

The most important event in head trauma is brain injury ,which may be :

a- Primary brain damage :at time of trauma,which may be focal or diffuse.

b- Secondary brain damage: imposed after initial injury due to hypoxia, hypotension & high ICP.

The aim of management is to prevent secondary brain injury.

Management of head injury follows the same principles of management of multi-injured patients:

I- Pre-hospital care:

1- Maintain patent air way supporting the mandible & drawing the tongue forward.

2- Cardio-respiratory resuscitation if arrested.

3- Arrest any bleeding by compression.

4- Transportation of the patient as one bolck with special care to airway patency and stabilization of cervical spine with neck collar.

II-Hospital care :

1-Primary survey: to detect what is killing the patient. It should follow the ABCDE rule.

2-Resuscitation: It should go simultaneously with the primary survey in the ABCDE rule :

1-Airway: It is of outmost importance to ensure patent airway because the commonest cause of early death after head trauma is obstructed airway. How to ensure patent airway?

2-Breathing:

-Ensure that the patient breathes well.

3-Circulation:

-Stop external bleeding. In scalp wounds, bleeding is controlled by evertion of the edges with Allis forceps.

-Correct shock ,if present, with IV fluids and/or blood transfusion but avoid volume overload to avoid brain oedema.

4-Disability= Dysfuntion of CNS:

*Search for signs of localization.

*Examine motor power & reflexes.

*Detect the level of consciousness by: **Glasgow coma scale (GCS)**

Eye opening		Best verbal response		Best motor response	
Spontaneous.	4	Oriented.	5	Obeys commands.	6
To verbal command	3	Confused.	4	Localizes pain.	5
To pain.	2	Inapporpriate words.	3	Flexion withdrawal.	4
None .	1	Incomprehensible sounds.	2	Abnormal flexion (decorticated).	3
		None.	1	Extension (Decerbrated).	2
				None .	1

The total score is 15. If is blow 8, the prognosis is bad.

5-Exposure & overall review:

-It is mandatory for perfect examination and detection of hidden injuries

3- Secondary survey: to detect other possible hidden injuries:

A- History: from the patient if s/he is conscious or from relevants. Ask about SAMPLE .

B- Examination: It is a head to toe evaluation:

-Head & Neck: Search for

* scalp wound & more importantly haematoma.

*bleeding orifices: nose, ear, mouth & cranial nerves injury (Q. fracture base).

-Examine thorax, abdomen, pelvis, limbs & spine for possible injuries.

Adjuncts to the secondary survey:

Investigations:

I-For head trauma:

1-Plain X-ray: fracture vault.

2-CT scan & MRI: of choice if available.

3-Carotid angiography: may help localization of haematoma.

4-lumbar puncture: if subarachinoid hage. is suspected , only few mls of CSF are drawn to avoid conization of the medulla into foramen magnum.

II For other systems:

Chest X ray, abdomional U/S, X ray for limbs, IVU- etc.

4- Definitive treatment:

I-*Conservative treatment* = Care of comatosed patient (for most cases):

1-Care of respiration

a) Ensure patent air *(see before).*

b) If spontaneous respiration doesn't keep normal level of PO_2 & PCO_2, endotracheal intubation & artificial ventilation are started. If they are needed for more than 7 days,. Endotrocheal tube is replaced with tracheostomy.

c) Mechanical suction of secretions.

2-Care of Bowel:

a) Nasogastric feeding. b) Daily enema.

3-Care of bladder:

Foley's urinary catheter to avoid irritability due to full bladder.

4- Control of pyrexia:

By cold fomentation & aspirin.

5- Control of convulsion:

- By chloral hydrate or chloropromazine. Barbiturates is contraindicated.

6- Control of brain oedema:

a) Mg. sulphate retention enema /12 hours. b) Frusemide.

c) Mannitol 20% 200 cc over 20 min twice daily. d)corticosteroids.

7-Frequent change of position to avoid bed sore.

8-Frequent estimation of bl. electrolytes.

9-Frequent observation and neurological examination to assess progress & prognosis.

II *Surgical treatment* : it is indicated in:

1-scalp wounds.

2-skull fracture when: a) compound fracture .

b) depressed fracture more than 3 mm in depth .

c) depressed fracture over motor or speech area .

d) pond fracture of the forehead which didn't correct itself in 6 weeks .

3-intracranial haemorrhage (extradural or subdural) mention the steps

4-deterioration after improvement

5- late indications to drain an abscess or reconstruct a bony defect of the skull by bone graft or metallic plates.

Intracranial infection

1- Brain abcess

Aetiology

I-Causative organisms ,wide variety of organisms are responsible but anaerobes are the majority.Fungi also may cause brain abcess

II-Route of spread

a-Direct spread:

-Direct spread from nearby infection such as otitis media frontal sinusitis, osteomylities of the skull. It is the comminest route .

- Direct implantation through compound fracture of the skull especially if foreign bodies are implanted

b- Haematogenous spread (not common)

from near or distant infection such as dental or tonsillar abcess,suppurative lung disease .it is usually multiple

III- Predisposing factors

-Immunocompromised patients e.g D.M, AIDS, and patient on immunosuppressive therapy

Pathology:

Brain abcess passes through the following stages.

1- stage of cerebritis which is characterized by marked exudation, inflammatory cell infilteration of the affected area

2- stage of localization : liquefaction occurs in the centre with gliosis occurs at periphery.

Complicatios:

1-Increased intracranial tension (ICT).

2-Damage to important centries in the brain.

3-Toxaemia and dissemination.

4-Fatal conization.

Cinical picture:

The manifestations of brain abscess are:

I-constitutional manifestation : fever is usually mild and may be absent.

II- manifestations of increased ICT ,headache , vomiting and blurred vision.

III-focal manifestations according to the site of the abcess e.g aphasia, hemiplegia, homonymous hemianopia .....etc

Investigations:

I-Laboratoty :

-leucocytosis and high ESR are usually evident.

N.B lumbar puncture to obtain CSF sample is contraindicated to avoid fatal conization

II-Imaging:

1-CT scan

2- MRI with gadolinium enhancement is the investigation of choice.

Differential diagnosis :

- Solitary abcess is difficult to be differentiated from solitary metastasis or tuberculomata

Treatment:

Treatment of brain abcess has three options:

I- Conservative treatment :

- It is indicated in - small sized < 1.5 cm in diameter
  -patient with bad general condition
- It includes - broad spectrum antibiotics
  -corticsteroid to reduce brain oedema
  -improving general condition and control predisposing factors.

II-Aspiration:

It is indicated mainly in 1-multiple abscesses
2-deeply seated abscess
3-abscess in critical location (near motor or speech area)
4-patient unfit for surgery

It is done through a burr hole in the skull with blunt ended needle under steriotactic image.

III-Surgical drainage :

It is indicated mainly in 1- multilocular abscess
2- superfacial abscess
3- Abscess on the top of foreign body
4- fungal abscess as it responds poorly to antifungal therapy

2-Extradural abcess

- It is commonly due to osteomylities of the skull less commonly due to extension from nearby infection or nasal sinuses
- The dura resists infection for a long time before the subdural space is infected (subdural abcess) The overlying scalp becomes markedly swollen and tender forming what is called *Pott's puffy tumour* (false tumour)
- treatment is by surgical drainage under umberlla of antibiotic with treatment of underlying cause

3-Subdural abcess

- It is usually due to extension from ear or nasal sinuses, rarely due to blood born infection
- It is characterized by severe toxaemia, progressive deterioration of consciousness & focal manifestations.Oedema of the overlying scalp is also evident.
- Treatment is by surgical drainage under umberlla of antibiotic through multiple burr holes and repeated aspiration of the subdural space.

Intracranial tumours

This term includes all benign and malignant neoplasms arising from meninges, brain tissue, cranial nerves ,blood vessels, pituitary and pineal glands or embryonic rests as well as secondaries

Pathology :

Types: intracranial tumours incudes

I - Primary tumours

a- from meninges: meningioma

b- from glial tissue: (glioma) the commonest primary

1- astrocytoma 2-oligodendroglioma

3- ependymoma 4-glioblastoma multiformis

c-from cranial nerves: acoustic neuroma

d-from blood vessels: haemangioma

e-from pituitary: Pituitary adenoma (functioning or non functioning)

f-from embryonic rests: 1-medulloblastoma 2-craniopharyngioma (from Ranthk's pouch)

II- Secondaries:

Brain secondaries are twice commoner than the primary tumours .

Complications:

1-Increased intracranial tension, papilloedema and optic nerve atrophy

2- Local damage to vital centres in the brain

3-Acquired hydrocephalus due to obstruction to CSF circulation

4 Fatal conization in neglected cases

5-Distant metastasis

Clinical picture :

Manifestations of brain tumour is non-specific and resemble those of any space occupaying lesion. They include

I- Manifestations of increased ICT: Headache ,vomiting and blurred vision. Also epilepsy

II-Focal manifestations ,according to the site of tumours

1-In supratentorial tumours:

a-hemiplegia,epileptic fits if motor area is involved

b-aphasia if speech area is involved

c-hemianaesthesia if the sensory area is involved

d-homonymus hemianopia if optic radiation is involved in temporal lobe tumour

e-anosomia ,mentality and psychological changes in frontal lobe tumour

2-In infratentorial tumous:

a-vertigo and ataxia in cerebellar tumours

b-cranial nerve palsy from the fifth to the twelfith

III-Special manifestations:

-Endocrinal disturbance in functional tumours of the pituitary e.g. galactorrhea, amenorrhea, gigantism etc

Investigations:

1- Plain X ray of the skull: it may show

1- increased indentation of the inner table of the skull giving beaten silver appearance

2-erosion of the posterior clinoid process and widening of sella turcica in pituitary tumours

3-calcification of the tumour e.g in craniopharyngioma

4-hyperostosis of the skull in meningiomas

5- widening of skull formina in skull base tumour

2- CTand MRI:

CT with contrast and MRI are now the investigation of choice

3-Carotid angiography:It was used to localize cerebral tumour also vertebral angiography to localized cerebellar tumours

4-Electrocephalography (EEG): It may show abnormal electric activity of the tumour

5-Biopsy :biobsy should ideally be taken by an stereostatic surgery

Differential diagnosis

From other space occupying lesion of the brain especially brain abcess

Treatment:

The only hope of perminant cure is surgical resection

Preoperative preparation

1-treatment of brain oedema with manitol 20% dexstrose 25% or corticosteroid

2- If hydrocephalus has resulted, it is primarly decompressed with ventriclo-peritoneal shunt

3-improve the general condition of the patient regarding nutrition.

-Excision of brain tumour can be done through craniotomy or craniectomy. Pituitary tumours can be removed through the trans sphenoid route

-Recently, CT or MRI guided stereostctic surgery is done to excise or biopsy deep tumours

-Adjuvant therapy (radio and /or chemotherapy)are used for malignant tumours

Brain secondaries

- Brain secondaries are the commenst intracranial tumours
- 25% of all cancer victims have brain metastasis
- 50% of them are solitary
- 25% of them are suitable for suitable for surgical excision
- Brain secondaries are commonly from bronchogenic or breast cancer.
- The approach to brain metastasis is either radiotherapy alone or surgical resection followed by total brain irradiation
- Prerequisition for surgical resection of brain metastasis

  1-truly solitary tumour.

  2-limited systemic dissementation

  3-life expectancy more than 2 months

  4- accessible tumour

  5-patient fit for surgery

Otherwise brain radiotherapy is the only what can be done

The Spinal Cord

Congenital Anomalies

Spina bifida

Congenital defect of the neural arch of the vertebra duo to absence of spinous process with or without meningeal & cord herniation.

Spina bifida

Site: The commonest site in the lumboscral region.

Types

l-Spina bifida occulta :

-The cord & meninges are intact & in place.

-There may be a band of tissue called membrana reunions attaching the dura to the skin of the back

-As the growth of the vertebra is more rapid than the growth of the cord so with time the membrana reunions pulls on dura and may compress over the cauda causing neurological problem (tethered cord)..

Spina bifida occulta

ll Spina bifida manifesta:

A-Meningocele:

-Herniation of the dura without the cord through the defect .

-It is filled with CSF.

Meningocele

2-Myelocele .

-Herniation of the cord & nerves bared (with no covering).

-It is incompatible with life.

3-Meningomyelocele

-Herniation of the dura containing the cord & equina bathed with CSF

-Sometimes the central canal of the spinal cord is markedly dilated within the herniated cord (*syringomyelocele)* (incompatible with life).

Meningomyelocele

as encephalocele.

Meningocele, myelocele & meningomyelocele resemble their cranial variants in all features.

-Excision of the sac as early as possible to avoid rapid rupture & fatal menimgits.

-If hydrocephalus develops, it is treated with shunt operation.

Trauma

Traumatic paraplegia

Aetiology:

Trauma to the spine: The cord is injured due to:

1-Stretch at time of injury.

2-Crushing between the fractured vertebral body and the laminae of the dislocated vertebra.

3-Compression of the cord by displaced fragments or haematoma.

Pathology:

There are three types or grades of cord injury:

1-Cord concussion: temporary loss of function.

2-Cord contusion: which is usually partial but permanent & uncorrectable.

3-Cord transection: complete damage of a segment of the cord.

Complications:

Complications of paraplegia (see internal medicine).

Clinical picture:

There are three stages:

1-Stage of spinal shock:

- There is complete flaccid paralysis in both lower limbs following trauma.
- It lasts about 48 hours in concussion to several weeks in contusion.

2-Stage of reflex activity:

- Lower motor neuron lesion persists at the level of injury.
- Upper motor neuron lesion below the injury with spastic paralysis , exaggerated reflexes and abnormal reflexes e.g. . Mass reflex (spinal automatism).

3-Stage of complications:

Bed sore, urinary calculi infection, pneumonia ... etc

Investigations:

Plain X ray, CT & MRI of the spine.

Treatment:

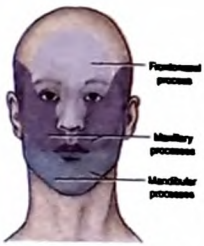
*Care of paraplegic patient (see internal medicine).

*Fracture & dislocation of the spine are treated as before.

-laminectomy & other decompression operation may be needed.

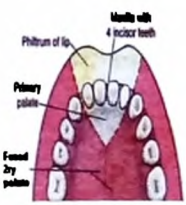
Face lip & palate

Embryology



Development of the face

- The face develops from five embryonic processes, frontonasal process, two maxillary processes & two mandibular processes.
- The frontonasal process forms the nose, the philtrum (median part of the upper lip), and premaxilla (the median part of the plate which is triangular. Its apex is in the incisive fossa and its base carries the incisor teeth.
- The two maxillary processes form the lateral parts of each upper lip and from each process; a transverse process called palatal process grows medially to meet its other fellow and a third median sagittal process called nasal septum in the midline. Lastly, the oral cavity is separate from the nasal cavity and both nasal cavities are separate from each other.
- The two mandibular processes fuse in the midline to form the mandible and the lower lip.
- The ear auricle is formed from the 2nd branchial arch by fusion of six auricular tubercles.



Development of the palate

I–Congenital Anomalies

A- Anomalies of fusion

1- Facial clefts:

1- Craniofacial Clefts:

- Between the frontonasal process & maxillary process.
- Unilateral or bilateral, complete or incomplete.

2- Cleft lip
3- Cleft palate
} Common (1/700 living birth).

Craniofacial cleft

2- Preauricular sinus:

- It is due to imperfect fusion of the auricular tubercles.
- If obstruction occurred, infection and abscess may ensue leading to persistent ulcer.

3- Macrostomia:

It is due to under fusion between the two maxillary and the two mandibular processes.

4- Microstomia:

It is due to overfusion of the maxillary & the mandibular processes.

5- Dermoid cyst: (sequestration dermoid):

It is common at the outer canthus (external angular dermoid).

B- Miscellaneous

1- Microtia: hypoplasia of the ear pinna.

2- Macrotia (bat ear): large ear auricle protruding laterally.

3- Micrognathia: receding mandible.

4- Macrognathia (prognathism): protruding mandible.

5- Macrocheilia: large lip due to cavernous haemangioma or lymphangioma.

Cleft lip (Hare lip) (شفة أرنبية)

Aetiology:

It is a congenital disease due to failure of fusion of the median process of the frontonasal process with one or both maxillary processes.

Predisposing factors:

1- Familial tendency: positive family history & more in consanguinity (قرابة دم)

2- Teratogenicity by Drugs: anticonvulsants

3-Irradiation: X-ray

4-Diseases: German measles

Pathology:

Site: - Unilateral > bilateral - Lt. > Rt.

- Rarely median (bifid or absent philtrum)

- Very rarely the lower lip

Unilateral incomplete

bilateral incomplete

Types:

1-Unilateral or bilateral

2-Complete or incomplete (reaches the nostril or not)

3-Simple or alveolar (associated with cleft palate)

simple complete

alveolar complete

Complications:

1- Disfigurement

2- Psychological upsets of the parents.

3- It may cause abnormal teeth growth.

4-It may interfere with labial letters e.g. M. P.

5- It does not interfere markedly with suckling.

Median lower lip

Median upper lip

Treatment:

Operation is best performed at the age of 3 to 6 months. The role of 10 is helpful

"About 10 weeks after birth, when weight is about 10 pounds and Hb is at least 10 gm % "

Aim: to improve the appearance and complications

Procedure: Many procedures are described. The most popular is *Milard's operation*

Stepss

1. Release the lip in the gingivo-labial sulcus to help approximation
2. Paring the edge to create raw areas
3. Design the flaps as shown
4. Suturing in three layers

Milard's operation

Mucosa to mucosa muscle to muscle skin to skin

N B – If cleft lip is associated with cleft palate, lip is repaired first to

1- Improve appearance and hence the psychological state of the parent

2- Avoid protrusion of the pre maxilla

Cleft palate

Aetiology

The same as cleft lip

Pathology

Types

1- Clefts of the 1ry palate alone

It may be unilateral or bilateral

2- Cleft of the 2ry palate alone

It may be bifid uvula, cleft soft palate or cleft soft & hard palate (intermaxillary cleft)

3- Cleft of the 1ry & 2ry palates:

It may be bipartite or tripartite

Unilateral **Bilateral**

Clefts of the primary palate

Cleft soft palate **cleft soft & hard palate**

Clefts of the secondary palate

Bipartite **Tripartite**

Cleft of the primary & secondary palate

Complications:

1- It interferes with normal suckling due to failure to create a negative intra-oral pressure

2- Regurgitation of food into the nasal cavity leading to

a) Nasal sinusitis

b) Aspiration pneumonia c) Otitis media

3- Hearing defect: due to inadequate aeration of the middle ear by abnormal insertion of the levator & recurrent otitis media

4- Speech defects

a – Inability to produce consonant letters e.g. K, D, P, T

b- Development of nasal tone, which is irreversible

5- Distortion of facial & teeth growth

Treatment:

Operation is best performed at the age of 12- 18 month (when begins to speak). Because it is bloody operation a newborn cannot withstand. However, the operation should be before the age 2 years (before development of irreversible nasal tone)

Aim

To correct the oronasal communication and its complications

Procedure: The most popular is Wardill operation.

Steps:

Repair with pushback technique is most popular

1- Paring the edges

2- Creation of mucoperiosteal flaps with preservation of the neurovascular pedicle (greater palatine artery & nerve)

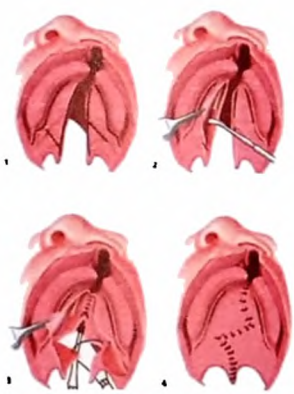
3- Detach the abnormal attachment of the levator palati muscle

4- Fracture the pterygoid hamulus to weaken the tensor palati muscle

5- Suturing the flaps in the midline in 3 layers

1- nasal mucosa

2- Mucoperiosteal flaps (platal mucosa).



Repair with push back method

With pushback technique to lengthen the soft palate in order to obtain competent velo-pharyngeal sphincter

6-Pharyngeoplasty: by transverse incision closed longitudinally or by purse string suture of the plate & pharyngeal wall. It is used in cases with incompetent velopharyngeal sphincter

II- Ulcers

1- Ulcers of the face

Inflammatory	Neoplastic
1- Pyogenic granuloma	1- Rodent ulcer
2- Ulcerating carbuncle	2- Squamous cell carcinoma
3- Keratoacanthoma	3- Ulcerating melanoma
4- Oriental (Baghdad) sore	4- Marjolin's ulcer
5- Lupus vulgaris (TB of the skin)	5- Cock's peculiar tumour (false tumour)

2- Ulcers of the lip

1- Cracked Lip

-It is a superficial painful ulcer, which occurs, commonly in cold weather

-It is always in the midline of the lower lip

-It bleeds easily and may cause marked inflammatory swelling of lip

-Treated with 1% silver nitrate painting but in resistant cases, excisional biopsy should be done

2-Herpetic ulcer (Herpes simplex)

-It is caused by herpes virus that usually follows common cold

-It is usually unilateral

-It occurs at the mucocutaneous junction but affecting skin > mucous membrane

-It starts as vesicles that rupture → ulcers

-It usually heals spontaneously but healing may be accelerated by topical antiviral agents.

3-T.B ulcer

-It is rare in the lip

-It has a pale granulation tissue floor, undermined edge and soft base

-It is very painful & the draining lymph nodes are usually enlarged

-It is treated by the classic anti T.B drugs, but excision & biopsy may be needed

4-Syphilitic *($)* ulcers: -

a)-Ulcerated (Hunter's) chancre (primary$)

-History of contact with infected partner

-It occurs on the upper lip

-It affects the skin > mucous membrane

-It has an indurated base and the draining LNs are always enlarged

B-Mucous patches (secondary $)

-It affects the mucous membrane

-Long shallow (snail track) ulcer spreading over the mouth and pharynx may appear

-Other manifestations of *secondary $* e.g. generalized lymphadenopathy

C-Gumnatous ulcer (tertiary $)

-Punched out painless ulcers with pale granulation tissue floor

-Regional LNs are not enlarged

5-Malignant ulcers

See below

III- Neoplasms

Carcinoma of the lips

Aetiology:

1-Predisposing factors: (5 Ss)

1-Sepsis 2- Spirits 3- Smoking (pipe) 4-Spices 5- Sun rays or radiation

2-Precancerous lesions: (5)

1-Papillomas 2-leukoplakia 3- lupus vulgaris T.B 4-$ 5- & burn

Pathology:

Site: lower lip 93% upper lip 5% angle 2%

MAC: There are 5 Types

1-Malignant ulcer (commonest) 2)malignant nodule

3)raised mucous plaque 4-malignant fissure

5-diffuse infiltrating type (woody lip)

MIC: Sq. cell carcinoma (describe)

Spread:

1-Direct spread to the rest of the lip

2-Lymphatic: characteristically by embolization

-Central part of the lip → submental LNs →

-Lateral part of the lip → submandibular LNs →

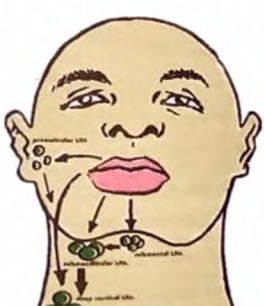
- Lat. part of the upper lip →preauricular LNs →

-Angle of the mouth →

Deep Cervical LNs

Lymphatic spread is slow

3-Bl. spread: is very rare & late



Lymphatic spread

Complications:

1-Bleeding 2-infection 3-dysarthria

Incidence:

Common in elderly male > 50 years

Clinical picture

The patient typically presents with chronic ulcer that has the following character

-*Site* : common in the lower lip - *size* : variable

-*Shape* : rounded oval or irregular - *edge* : raised everted

-*Floor* : necrotic - *base* : induration beyond the margin

-It may extend to the cheek and may be fixed to gum or mandible

-*Cervical L.N.s* may be enlarged (rare) either due to infection (firm & painful) or due to metastasis (hard, painless & may be fixed)

Investigations:

Excisional biopsy

Differential Diagnosis:

From other ulcers of the lip

Treatment:

Most cases are operable

1- Surgery: wide excision with ½-inch margin

Indications

1)Early cases (not more than 1/3 of the lip) 2)Late cases infiltrating bone

3)Radioresistant tumors 4) Radiorecurrent tumours

5)Carcinoma on top of *S*

-Lip reconstruction may be primary in small tumours or with the aid of flaps e.g. cross lip flap in large tumours

-The most important cosmetic point is to preserve the alignment of the red margin

2- Radiotherapy: using radium infiltration needles of 7000 Rads

Prognosis:

Epithelioma of the lip has a better prognosis than that elsewhere in the face or oral cavity

The Mouth, cheek & Tongue

I- Congenital anomalies

1- Aglossia: due to arrested growth of tuberculum impar.

2- Bifid tongue: due to failure of fusion between the two lingual processes.

3- Ankyloglossia (tongue-tie)**:** due to short & thick fraenum linguae.

-It may cause swallowing & speech disorder.

-It should be treated by division of the fraenum near the floor of the mouth.

4- Lingual thyroid: It presents as a red lobulated mass behind foramen caecum.

5- Macroclossia: congenital macroglossia is due to:

1-Cavernous haemangioma. 2- Cavernous lymphangioma.

3-Congenital A-V. fistula. 4- Congenital neurofibroma. 5-Cretinism.

II- Cysts

Cysts in the floor of the mouth

1-Ranula: (ranula = frog)

Aetiology:

It is a retention cyst or extravasation cyst of sublingual mucous gland.

Pathology:

Site: in the floor of the mouth on one side of the tongue pushing it to the other side (*simple ranula*). It may dissect its way behind the posterior border of the mylohyoid muscle to appear in the neck (*plunging or dissecting ranula*).

Composition: -It is lined with columnar epith.

-It contains jelly like material rich in mucin but no salivary enzymes.

Clinical picture:

- Usually in infancy or early childhood.
- Bluish translucent cystic swelling with obvious blood vessels over it pushing the tongue to one side.
- It may cross the midline and become constricted by the fraenum linguae giving an hourglass appearance.
- It may cause difficulty in swallowing and speech.
- Plunging (dissecting) ranula may appear in the neck.

Ranula

Treatment:

1-Simple ranula: surgical excision is very difficult. Marsupilization is the treatment of choice by deroofing of the cyst and suturing the wall to the oral mucosa.

2-Plunging ranula: may need cervical incision for its dissection.

2-Sublingual dermoid:

Aetiology:

Sequestration dermoid which is formed during fusion of epithelium. on both halves of the tongue.

Clinical picture:

-*Supramyohyoid type:* opaque swelling in the floor of the mouth. It may be in the midline or lateral pushing the tongue to one side. It may cause dysphagia, dysarthria & dyspnea.

-*Inframylohyoid type :* swelling in the submental or digastric triangle. It gets more prominent during deglutition.

Treatment:

Surgical excision.

3-Mucous retention cyst:

- It is a retention cyst of buccal mucous gland.
- It appears as a small bluish translucent cyst anywhere in the buccal cavity.
- Surgical excision can be done under local anaesthesia.

4-Suprahyoid thyroglossal cyst:

5-Cystic tumours: e.g. haemangioma, & lymphangioma.

III- Ulcers

Ulcers of the tongue

1-Dyspeptic ulcers: the commonest (Aphthous = mouth ulcer):

- They occur near the tip and may be multiple.
- They are small, rounded, & shallow with yellow floor & soft base. The margin is hyperemic.
- They are very painful.
- They heal spontaneously within 2 weeks. Healing may be hastened by antiseptic wash, alkaline wash as sodium bicarbonates 2%.

2-Traumatic ulcers:

a) Dental ulcer :

-It is due to repeated trauma by broken tooth or ill fitted denture.

-It occurs at the side of the tongue (near the accused tooth).

-It is small rounded or oval with sloping edge and soft base.

-The floor is covered with infected granulation with septic enlargement of draining LNs.

-It is painful ulcer and may turn malignant.

-It is treated by correction of the cause, antibiotics, antiseptic mouthwash & biopsy in ch. cases.

b) Post pertussis (fraenal) ulcer:

-It is due to repeated trauma of the fully protruded tongue during bouts of violent cough.

-It has the same characters and treatment of dental ulcer except that it occurs at the fraenum.

c) Post epileptic ulcer:

It is due to trauma (biting) of the tongue during epileptic fit.

3-Turberculous ulcer:

- It occurs on the tip or the back.
- Multiple and painful.
- Small round or oval ulcers with undermined edge and pale granulation tissue floor.
- It is painful with enlarged draining LNs.
- Treatment is by antituberculous drugs

4-Syphilitic ulcers:

-The tongue may be affected in the three stages of syphilis:

a) 1ry *S*: chancre occurs at the tip with enlarged submental LNs.

b) 2ry *S*: mucous patches with snail track ulcers.

c) 3ry *S*: - Gummatous ulcer occurs in the midline on the dorsum

- It is painless, punched out ulcer.

Ulcers of the tongue

5-Malignant ulcer (see later)

-It is more common in old males.

-It is common on the sides of the anterior two third.

-It has a raised everted edge, necrotic floor & indurated base.

IV- Inflammation

I- Acute Glossitis:

1- Acute superficial glossitis

2- Acute parenchymatous glossitis

II- Chronic glossitis

1- Chronic superficial glossitis:

2- Tuberculous glossitis

3- Syphylitic glossitis

V-Neoplasms

Carcinoma of the tongue

(Cancer of 5)

Aetiology:

I-Predisposing factors: (5Ss)

: Sepsis, Spirits, Smoking, Spices & Sharp tooth.

II Precancerous lesions :(5)

1- Leukoplakia. 2- Dental ulcer. 3- *S* ulcer

4- Papilloma 5- Ch. Superficial glossitis.

Pathology:

Site: -in the sides of the anterior 2/3 (commonest)

- on the post .1/3 (less common)

- on the ventral & dorsal surfaces or the tip (least common).

MAC: (5) macroscopic types

1-Malignant ulcer (commonest) mention characters. 2-Malignant nodule.

3-Raised oval plaque. 4-Malignant fissure.

5-Diffuse infiltrating type (woody tongue).

MIC: (5) microscopic types

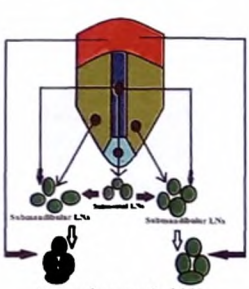
1-Squamous cell carcinoma (commonest).

2-Lymphoepithelioma (carcinosarcoma) (post 1/3).

3-Basal cell carcinoma.

4-Adenocarcinoma (minor salivary glands).

5-Pure sarcoma.



Lymphatic spread

Spread:

1-Direct spread:

Anterior 2/3: gum, floor of the mouth, & mandible.

Posterior 1/3: tonsils, pharynx, larynx and soft palate.

2- Lymphatic spread: (early)

-From the side → ipsilateral submandibular LNs → ipsilateral upper deep cervical LNs .

-From the tip of the tongue → submental LNs → bilateral deep cervical LNs.

-From the central part & from the posterior 1/3 → bilateral deep cervical LNs.

3- Blood spread: rare except in the posterior 1/3.

Complications: **5**

1- Infection: is common leading to foul odour and oedema that may be risky.

2- Respiratory complications:

- Asphyxia due to marked oedema or inhalation of sloughed tissue.
- Inhalation bronchopneumonia.

3. Haemorrhage: due to erosion of major blood vessels e.g. the lingual artery.

4. Starvation & weight loss.

5. Dysphagia, dysarthria.

Incidence:-

Common in old males > 50 years.

Clinical picture: **5** ***presentations***

1-Pain: early due to infection and late due to infiltration of the lingual nerve. Pain may be referred to the ear through the chorda tympani nerve.

2- Profuse salivation: due to pain and inability to swallow.

3-Anklyoglossia: inability to protrude the tongue with deviation to the affected side due to infiltration of the muscles.

4- Mass: either the primary in the tongue or secondaries in the neck.

5- The patient frequently presents with complications, e.g. dysarthria, dysphagia, infection with fetor oris, asphyxia or respiratory infection.

Investigations: **5**

1- Biopsy from the edge of the ulcer.

2- X ray of the mandible.

3- FNAC of the suspected cervical LN(s).

4- CT of the neck & mandible.

Typical malignant ulcer

5- Preoperative investigations to detect fitness for operation.

Differential Diagnosis:

From other swellings of the tongue (5) & other ulcers of the tongue (5).

Treatment:

1- Radiotherapy: is indicated in

1. Small early lesion (< 2 cm) as alternative to surgery (optional).
2. Tumours of the post 1/3.
3. Unfit patient.
4. Inoperable cases.
5. Recurrence after surgery .

It can be performed by Cesium or iridium needles or by external beam radiotherapy.

2- Surgery:

Indications (like cancer lip)

1-Very early tumors
2- late tumours with bone infiltration
3- Radioresistant tumour
4- Radiorecurrent tumour
5- Carcinoma on top of syphilis

Aim: To remove the tumour with 1 inch safety margin together with block neck dissection

Procedures:

One of the following according to the size

1- Partial glossectomy.
2- Hemiglossectomy.
3- Near total glossectomy.
4- Total glossectomy.
5- Glossectomy + Commando (Combined mandiblectomy and neck dissection operation) for tumours infiltrating the bone.

- Block neck dissection of the cervical LNs should be performed on one or both sides whether the glands are palpable or not (lymphatic spread is early & common).
- It is better carried out 2 weeks after healing of the primary to allow malignant cells that would be in the lymphatics to settle down in the lymph nodes.

Prognosis:

The prognosis depends on many factors:

1- Lymphatic spread is the most important.
2- Staging.
3- The differentiation of the tumour.
4- Site of the tumour worst in the post . 1/3.
5-Sex: prognosis is worse in males.

The 5-year survival rate is about **25%**.

Swellings of the tongue

1-Retention mucous cyst: -Small painless rounded cystic swelling with smooth surface.

2-Haemangioma & lymphangioma.

3-Benign tumors

1-papilloma. wart like mass which maybe sessile or pedunculated.

2-fibroma: firm, pedunculated.

3-Neurofibroma may be single or multiple

4-Malignant tunours: See below

5-Macroglossia: See before

Gum, Teeth & Jaws

Swellings of the gum (epulis)

1) Granulomatous epulis:

Granulomatous epulis

- It is a mass of granulation tissue adjacent to a carious tooth.
- It is a small lobulated mass devoid of epithelial covering.
- It is red and bleeds easily.
- Treatment: Extraction of the carious tooth with excision of the granulation tissue with diathermy.

2) Fibrous epulis:

Fibrous epulis

- It is a true fibroma of the gum (mucoperiosteum of the jaw).
- It usually arises near the incisor teeth of the lower jaw.
- It is a small painless pedunculated swelling that does not cause wide separation of the teeth.
- It is whitish pink (less vascular) and does not bleed easily.
- *Treatment:* Extraction of the teeth on both sides then, wide excision of the tumor with wide safety margin.

3) Myeloid epulis:

Myeloid epulis

- It is osteoclastoma of the gum.
- It usually arises in the lower jaw.
- It is painless lobulated sessile mass causing wide separation & loosening of teeth.
- It is purple (vascular) and may cause serious bleeding.
- Treatment: Wide excision with safety margin e.g. Hemimandibulectomy.

4) Sarcomatous epulis:

- It is a parosteal fibrosarcoma of the gum (many arise on top of fibrous epulis). "Parosteal = adjacent to the periosteum"
- It is a sessile mass fixed to the bone.

Treatment: Wide excision with safety margin e.g. Hemimandibulectomy.

5) Carcinomatous epulis:

- It is an epithelioma of the mucous membrane of the gum.
- It forms an ulcerated infected mass with raised everted edge, & necrotic indurated floor

Treatment: Surgical excision with safety margin and mandibular reconstruction. Radiotherapy has no role.

Swellings of the teeth remnants (Odontomes)

Definition:

They are cysts or cystic tumors arising from dental structures. *They are normal dental tissue that has grown in an irregular way)*

Types:

1. From epithelial remnants: - Dental cyst.
   - Dentigerous cyst. (Gerous means "bearing," "producing,")
   - Admantinoma. (Greek word adamantinos, means "very hard ")
2. from connective tissue: e.g. cementoma (rare in man). "Cementum is a thin layer of bony material that fixes teeth to the jaw."
3. Composite Odontomes.

1- Dental Cyst

Aetiology:

It is due to chronic irritation of paradental epithelial debris of Malassez by a nearby-infected tooth (usually the incisor or canine).

Pathology:

Site: it is common in the upper jaw especially anterior part.

MAC: it is mall unilocular cyst, slowly growing, expanding the jaw on both sides (inward & outward).

MIC: it is lined by squamous epithelium and contains mucoid fluid rich in cholesterol.

Dental Cyst

Incidence:

Common in adult life.

Clinical picture:

- It is a small, painless, slowly growing swelling in the anterior part of the upper jaw.
- It may give eggshell crackling sensation (thinned bone).

Investigations:

- Plain X-ray shows expansion of the jaw with defined cyst that is empty.

Treatment:

Extraction of the infected tooth and opening the cyst through incision in the gum and curettage of its contents followed by crushing of the alveolus.

2- Dentigerous cyst

Aetiology:

It is due to cystic degeneration of a dental follicle.

Dentgerous cyst

Pathology:

Site: It commonly arises in the lower jaw in relation to an unerupted tooth (the 3rd molar).

Composition:

- The cyst contains a maldeveloped maldirected tooth & filled with viscid fluid.
- It grows to outside causing thinning out of the outer table.
- There are ridges in its wall (pseudotrabeculae).

Incidence:

It is common in children & adolescents in the age of second dentition (7-25 years).

Clinical picture:

- Small, painless, slowly growing swelling near the angle of the lower jaw.
- It may give eggshell crackling sensation.
- There is usually missed (unerupted) tooth.

Investigations:

Plain X ray: It shows the expansion of the jaw with a cyst containing maldirected tooth and shows *"soap bubble appearance"* due to pseudotrabeculae.

Treatment:

- Deroofing of the cyst, removing the tooth, curettage the crushing the alveolus

3- Admantinoma (Ameloblastoma)

Aetiology:

It is a basal cell carcinoma of the paradental epithelial debris of Malassez

Pathology

Site: It commonly arises in the lower jaw near the angle

Composition:

- Multilocular cyst with thick trabeculae & containing brownish mucous fluid
- It grows more to the outside with thinning out of the outer table
- It grows both forwards in the body and upwards in the ramus

Ameloblastoma

Complications:

1. Ulceration, infection.
2. Bleeding.
3. Falling of teeth & pathological fracture (rare).
4. Recurrence after local excision.
5. Malignant transformation.

ulceration

Incidence:

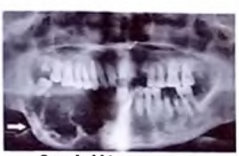
It is more common in young adult females (20 – 45 years).

Clinical picture:

- A painless, lobulated mass near the angle of the mandible.
- It is hard in consistency with possibility of egg shell crackling sensation.
- It may loosen teeth, and may ulcerate.
- Enlarged LNs are usually due to secondary infection.

Investigations:

Plain X-ray: It shows multilocular cyst with *soap bubble appearance* or *honeycomb appearance* with equal lobulations.



Soap bubbles appearance

***Differential diagnosis*:**

From osteoclastoma of the mandible which has the following characters:

	Admantinoma	Osteoclastoma
Incidence	-More common -Young adult females	-Less common -Younger age group (15-30)
Site	-Near the angle of the mandible	-Near the symphysis menti
Spread	-In both horizontal & vertical direction -It may cross the midline -It grows more to the outer side	-In horizontal direction only to stop short out of the angle -It never crosses the midline -It grows on both sides equally
X ray	Multilocular with equal loculation giving fine soap bubble appearance	Multilocular with unequal loculation giving fine & coarse soap bubble appearance

Treatment:

Resection of the affected segment of the mandible with a safety margin of at least ½ inch. The lost bone is replaced by a bone graft e.g. rib or artificial prosthesis.

True bone tumors of the jaw

1- **Ivory osteoma:** It arises from bones developed from membranes e.g. ramus of the mandible, outer surface of the maxilla & palate. It is treated by excision.

2- **Cancellous osteoma:** It arises from bones that develop from cartilage e.g. maxilla & symphysis menti. It is treated with excision (chiseling).

3- **Giant cell tumor (osteoclastoma):** It commonly affects the mandible near symphysis menti. Best treatment is resection of the mandible and reconstruction.

4- **Sarcoma:**

a) Osteosarcoma (osteogenic sarcoma): It is common in the upper jaw and may be on top of Paget's disease. Treatment is maxillectomy.

b) Fibrosarcoma (parosteal fibrosarcoma). It arises from the periosteum of the jaw.

c) Burkett's lymphoma.

5- **Secondaries:** They are usually secondaries from carcinoma of the oral cavity e.g. cancer lip, tongue & salivary glands, usually by direct spread (infiltration).

- It is treated by block dissection with the primary site (Commando).
- Radiotherapy has no role & better avoided as it may lead to necrosis of the mandible.

Carcinoma of the maxilla

Aetiology:

Cells of origin: the mucous membrane lining the antrum, palate, gum or teeth sockets.

Premalignant conditions: chronic maxillary sinusitis.

Pathology:

Site: It arises most commonly from the antrum and encroaches upon one or more of its walls.

Mac: It is usually of the fungating type.

Mic: It may be - adenocarcinoma (most common) if it arises from the antrum.
- Squamous cell carcinoma if it arises from the palate, gum or teeth sockets.

Spread: Direct spread: to the surrounding structures.
Lymphatic spread: to the upper cervical and retropharyngeal lymph nodes.
Blood spread: rare.

Complications:

1- Encroachment over the surrounding structures (see below).

2- Cosmetic disfigurement.

3- Distant metastasis

Incidence:

It affects both sexes equally specially above the age of 40 years.

Clinical picture:

- The earliest presentation is chronic sinusitis that is not responding to the ordinary treatment. In addition, it is suspected when nasal discharge is always bloody.
- Later, the presentation varies according to the wall of the antrum which is encroached upon:
  - Extension to the anterolateral wall leads to cheek mass that may fungate and ulcerate.
  - Extension to the medial wall leads to nasal obstruction, epistaxis and epiphora (nasolacrimal duct obstruction).
  - Extension to the roof leads to proptosis, diplopia & finally loss of vision.
  - Extension to the floor leads to depression of the hard palate and later fungation into mouth.
  - Extension to the posterior wall leads to obstruction of the nasopharynx & cranial nerve palsy.
- Enlarged cervical lymph nodes may be the earliest presentation.

Investigations:

1- Plain X ray: It shows opacity in the maxillary antrum with bone erosion.

2- CT & MRI: They detect the site, size & extent of the tumor. In addition, they detect LN metastasis.

3- Biopsy: -piecemeal from the mass.
- exfoliated cytology from antral lavage.
- FNAC from LNs.

Differential diagnosis:

From other solid swelling of the jaw (see later) especially sarcoma of the maxilla in which:

1- Younger age group is affected.

2- Rapid rate of growth leads to shorter duration.

3- Pain is early.

4- Early metastasis leads to worse general condition and worse prognosis.

Treatment:

I- Early cases: Either surgery or radiotherapy. Surgery is preferred in case of bone infiltration. Total maxillectomy and replacement with maxillary prosthesis to avoid facial deformity.
- Block neck dissection is performed only if the cervical LNs are involved.

II- Late cases: combined radiotherapy and surgery
- Intra cavitary radiotherapy may be used.

The Major Salivary glands

Anatomy

Anatomy of the parotid gland

- It is the largest salivary gland
- It is wedged between the mandibular ramus & the anterior border of sternocleidomastoid muscle below the ear auricle
- It is enclosed in a tough part of the deep cervical fascia that is named parotid fascia.
- It is pierced by the facial nerve from its posterior deep aspect which with its terminal branches divides the gland into two surgical (not anatomical) lobes; the superficial & deep lobes.
- The deep surface of the gland is related to:
  - One bone: styloid process.
  - Two arteries: the external and internal carotid arteries.
  - Three muscles: stylohyoid, stylopharyngeus and and styloglossus muscles.
  - Four nerves; the last four cranial nerves.

Surface anatomy of the parotid gland

- **Parotid duct** or ***Stensen's duct*** :-
  - It is about 5 cm in length.
  - It arises from the anterior aspect of the gland and passes over the masseter muscle within buccal pad of fat then it pierces the buccinator muscle to open in the vestibule of the mouth opposite the upper second molar tooth.

Surface anatomy of the parotid duct:

It is represented by the middle one third of a line extending from the tragus to the mid point between the ala of the nose & angle of the mouth.

- **The parotid gland is pierced by the following structures (from superficial to deep):**
  1- The facial nerve and its terminal branches.
  2- The retromandibular vein (the posterior facial vein).
  3- The external carotid artery: where it gives its two terminal branches , the maxillary and superficial temporal arteries.
  4- the auriculotemporal nerve.

The Terminal branches of facial nerve are:

The temporal, zygomatic , buccal , mandibular & cervical branches.

Blood supply

1- Arterial: from the external carotid & its terminal branches.

2- Venous: through the external jugular vein.

Nerve Supply:

1- Parasympathetic: through the auriculotemporal nerve.

2- Sympathetic: from sympathetic fibres around the external carotid art.

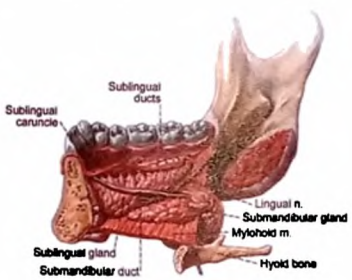
Branches of the facial N.

Anatomy of the submandibular gland

- It is formed of two parts:

1- The superficial part: is wedged between the body of the mandible and the mylohyoid muscle (superficial to the muscle).

2- The deep part: is the small part of the gland that hooks around the posterior border of the myolohyoid muscle to lie between it & the hyoglossus muscle (deep to the myolohyoid muscle) & between the lingual nerve superiorly & the hypoglossal nerve inferiorly.



Anatomy of the submandibular gland

The submandibular (Wharton's) duct:

It arises from the anterior end of the deep part and passes anteriorly to open on the side of fraenum lingae (It is about 5 cm) . It is hooked by the lingual nerve that passes superficial, below then deep to the duct .

Blood supply:

From the facial & lingual artery.

Nerve supply:

From the submandibular ganglion.

Anatomy of the sublingual salivary gland

It is the smallest of the three major salivary gland .It lies in the floor of the mouth , deep to the mandible & opens by multiple ducts into the sublingual fold.

***Minor salivary glands**. There are 800-1000 minor salivary glands located throughout the oral cavity within the submucosa of the buccal, labial, and lingual mucosa. Also, the soft palate, the lateral parts of the hard palate, and the floor of the mouth .They can be a source of salivary glands tumors.

Diseases of Salivary glands

I- Inflammation

A- Acute inflammation.

1- Acute epidemic parotitis:

Aetiology:

It is due to mumps or influenza viruses.

Pathology:

-It is commonly bilateral.

- The glands are enlarged and oedematous.

- It usually resolves completely with no residual damages.

Treatment: conservative.

2- Acute (bacterial) suppurative sialadenitis:

Aetiology:

I- Causative organisms: the commonest is Staph aureus then Streptococci, & Pneumococci.

II- Route of spread:

1- Along the duct from the oral cavity especially after operation due to dryness of mouth & lack of oral hygiene.

2- Blood born.

3- Direct from a nearby focus.

III – Predisposing factors:

1- Lack of oral hygiene.

2- Dryness of mouth postoperativly & in mouth breathers.

3- Obstruction of the duct by food particles or stones.

Pathology:

- The parotid is the commonest gland to be affected.

- The gland is swollen, congested & may suppurate.

Complications:

1- Toxaemia, septicaemia & pyaemia

2- Spread : to the surrounding . Submandibular sialadenitis may cause cellulitis in the submandibular region *" Ludwig's angina"* .

3- Abscess formation, which may rupture.

4- Salivary fistula.

5- Chronicity with stone formation.

Clinical Picture:

General: constitutional manifestations as fever, headache, anorexia & malaise.

Locally:

1- Pain: early dull aching but later, it is severe throbbing.

2- Swelling: oedematous gland.

3- Trismus : restricted movement of tempromandibular joint.

4- Red, oedematous opening of the parotid duct which may discharge pus.

5- Fluctuation is very late due to thick parotid fascia. The abscess may point to the pharynx.

Treatment:

I- Conservative tt:

1- Rest 2-Avoid sialagogues. 3- Antibiotic (antistaph).

4- Analgesic. 5- Hot fomentation.

This therapy is given for 48 hours provided there is no signs of suppuration.

II – Surgical tt:

- If conservative tt fails (no improvement).

-If there are signs of suppuration (throbbing pain, hectic fever & pitting oedema), surgical drainage is done

" Don't wait for fluctuation"

1- Parotid abscess:

Oblique incision in the skin and transverse incision in the parotid fascia " to avoid injury to the branches of facial nerve " then the abscess is opened by *Hilton's method.*

2- Submandibular abscess

An oblique incision parallel to the lower border of the mandible stopped 1½ cm below & infront of the angle of the mandible " to avoid injury to the mandibular branch of the facial nerve " then open the abscess by Hilton's method.

B- Chronic endemic parotitis:

Aetiology: it is usually associated with:

1- Parasitic infestation especially Ankylostoma.

2- Liver cirrhosis.

3- malnutrition.

Clinical picture

It is usually bilateral, soft, non-tender parotid swelling.

II- Calculi

Salivary Calculi

Aetiology:

I- Infection: - Infection is a good farmer for stones as it provides:

1- Nucleus: through the tissue debris & bacterial colonies.

2- Soil: ulceration of epithelium.

3- Climate: change the pH of saliva.

4- Manure: the exudate is sticky gluing the constituents together.

5- Time: It causes stasis that gives time for stone formation.

II – Obstruction by :

1- Stenosis. 2- F.B.

Pathology:

Site : -It may be in the gland or the duct .It is much more common in the submandibular gland than in the parotid (about 50 : 1) due to:

1- the secretion of the submandibular salivary gland is more viscid & tenacious.

2- the drainage of submandibular gland is independent.

3- Its duct opens in the floor of the mouth with possibility of obstruction by food particles.

Composition:

They are composed of Calcium , Magnesium, phosphates & carbonates.

Complications:

1-Migration: It causes salivary colic.

2-Obstruction: It causes chronic enlargement of the gland & predisposes for infection.

3-Infection: It may end in abscess formation.

4- Ulceration & bleeding 5- Fistula formation.

Clinical Picture:

1- Pain which may be :

- dull aching pain if the stone is in the gland or colicky pain (salivary colic) if it is in the duct.It increases with meal & mastication with referral to the tip of the tongue.

Stone in submandibular duct

2- Swelling:

- The gland is enlarged , tender & its size is increased after meal.
- Stone in submandibular duct may be felt through the floor of the mouth.

3- Discharge: Bloody or even purulent discharge may be seen coming from the duct.

4- Picture of complications.

Investigations:

1- Plain X ray film:

The stones are almost always radio-opaque but, they may be hidden by the mandible. This obstacle is overcome by:

- Radiographing in mild tilt of the head.
- Radiographing while the patient is pushing his mouth floor down with his index.
- Dental film (small film is clenched between the two jaws and X ray is emitted from below.

X ray film lat. veiw with head tilt

2-Sialography:

A radio-opaque material (lipidol) is injected in the canulated duct to delineate it and show the so rare radiotranslucent stones.

Treatment:

1-Stone in the duct: It may be easily removed through small incision in the duct from the floor of the mouth. The incision is left open.

-If this is not possible or recurrent stone → submandibular sialadenctomy.

2-Stone in the glands: submandibular sialadenctomy.

Salivary fistula

Aetiology:

1-Trauma: stab or operation.

2-Abscess: ruptures or incised.

3-Salivary stone.

4-Malignant tumour.

Pathology:

Site: -It may be in the duct (usually traumatic) or in the gland (usually follows suppuration).

-It is more common in the parotid duct.

Types: It may be *1-internal*: opening in the mouth (no harm).

2-External: opening into the skin.

Clinical picture:

-History of the cause.

-An abnormal opening (sinus) on the skin discharging saliva with eczema of the skin around.

The discharge increases with meals & mastication.

Investigations:

-Sialography to show the type & site of fistula.

Differential diagnosis

From other sinuses or fistulae of the face:

1-TB sinus.

2 -actinomycosis.

3-sinus of ch. osteomyelitis of the mandible.

4-preauricular fistula.

Treatment:

1-Fistula in the gland (parotid)

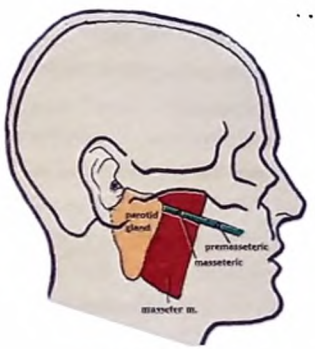
Suppression of secretion by one of the following:-

1-Atropine or its derivatives.

2-Avulsion of auriculotemporal nerve.

3-Irradiation of the gland.

If this fails resection of the gland is indicated.



Parotid fistula

2-Fistula in the duct (parotid):

1-Premasseteric fistula: (The opening is anterior to the anterior border of the masseter muscle). It could be treated by splitting the duct in the inner side of the cheek from its opening to the level of the fistula to create a wide internal fistula ,so the external one closes.

2-Masseteric fistula: the opening lies over the masseter muscle:

-It is difficult to treat.

-The ideal treatment is microsurgical anastomosis of the two ends over a wire splint.

-If it fails, inhibition of parotid secretion is tried as before.

III- Neoplasms

Benign tumours	Malignant tumours
1-Pleopmorphic adenoma	1-Pleomorphic adenocarcinoma
2-Adenolymphoma (Warthin tumour)	2-Adenocarcinoma
3-Oncocytoma (oxyphil adenoma)	3-Adenoid cystic carcinoma "cylindroma)
4-Monomorphic adenoma	4-Mucoepidermoid carcinoma
5-Haemangioma & lymphangioma	5-Epidermoid carcinoma

A- Benign tumours

1- Pleomorphic adenoma
(Benign mixed tumour)

Pathology:

Site: It is common in the parotid gland.

MAC: small firm mass with lobulated surface & grayish white cut section.

MIC: There is *pleomorphism*:

-The cells are spindle or stellate shaped and arranged in acini, clusters or sheets.

-The stroma appears blue resembling that of a cartilage and hence the old name (benign mixed tumour). Now, it is proved that this blue material is a mucinous secretion from the cells.

-There are microscopic extensions beyond the capsule of the tumour and this is responsible for high incidence of recurrence after simple enucleation.

-It is considered a locally malignant tumour.

Complications:

1-Cosmetic disfigurement.

2-Malignant changes are rare (3%) & late (after many years).

Incidence:

-It is the commonest salivary tumour (about 70 % of all benign salivary tumours).

-It is common between (30-50) years and equal in both sexes.

Clinical picture:

Slowly growing swelling which has the following characters:

-Single.

-Site : usually in parotid region. - -Size . small.

-Shape : hemispherical. -Surface : smooth lobulated.

-Skin over: not attached to the tumour. -Surrounding structures: *Facial nerve is free.

*Masseter muscle is free.

-Special characters:

*It becomes more prominent on contraction of the masseter.

*It becomes less prominent on opening of the mouth due to stretch of parotid fascia.

N.B. Parotid swellings have the following characters
-They fill the space between the ramus of the mandible & the mastoid process.
-They may raise the lobule of the ear.
-They become more prominent on contraction of masseter muscle
-They become less prominent on opening of the mouth

Investigations:

1-Fine needle aspiration cytology: It is safe and with high specificity ,but needs expert cytologist.

2- Open biopsy is absolutely contraindicated except with fungation and ulceration.

3-Isotope scan: TCm ^{99} scan.

Most salivary tumours give cold spots. Warthin's tumour & oncocytoma give hot spots.

4-CT& MRI scans: Helpful in deep tumors and give accurate information about tumor confines.

Differential diagnosis:

From other swellings in the parotid region:

I-Swelling of the parotid gland, (They have the previous characters)

1-Inflammatory: Acute viral, Acute bacterial, endemic & chronic parotitis.

2-Calcular: Enlarged gland with stone in the duct.

3-Auto-immune: Miculicz's & Sjogren's sydrome.

4-Drug induced enlargement of the parotid: Sulfa, Iodide containing drugs, phenylbulazone & contraceptive pills.

5-Neoplastic: Benign & malignant neoplasms.

II Extraparotid swellings (General swellings)

1-From skin: Sebaceous cyst, & abscess.

2- S.C tissue: lipoma.

3- LNs: preauricular & parotid LNs.

4-Muscle: Hypertrophy of masseter muscle: It usually occurs in young ladies who have the habit of grinding their teeth (*bruxism)* and it is bilateral.

5-Bone: Tumour of the mandible or maxilla.

Treatment:

1-Enucleation: is followed by high incidence of recurrence.

2-Superficial conservative parotidectomy:

If it is in the superficial lobe, the facial nerve should be explored with its branches and preserved.

3-Total conservative parotidectomy:

It is indicated in tumours of the deep lobe & in recurrent cases.

The whole gland is removed with preservation of facial nerve.

Y shaped incision

Lazy S incision

2-Warthin tumour

(Papillary cystadenoma lymphomatosum - Adenolymphoma)

Aetiology:

It is thought that it originates from heterotopic salivary tissue within parotid LNs.

Pathology:

Site: in the lower part of parotid gland. It is bilateral in 10% of cases.

MAC: It is small cystic swelling, which is well encapsulated.

MIC: The tumour is formed of multiple cystic spaces with multiple finger like papillae projecting into them and surrounded with lymphoid tissue.

Incidence:

-It represents about 10% of all tumours.

-It affects males over 50 years.

Clinical picture:

-It is represented by a small slowly growing swelling in the lower parotid with very soft or even cystic consistency.

Investigations:

-Isotope scan: unlike most of salivary tumours, Warthin's tumour gives hot spot in isotpe scan.

Treatment:

Local excision and recurrence doesn't occur (well encapsulated).

B- Malignant tumours

Pathology:

Site: They are common in the parotid. However, the percent incidence of malignancy varies inversely with the size of the gland i. e. malignant tumours constitute 25% of parotid tumours, 40 % of submandibular tumours & 70% of sublingual & minor salivary glands tumours.

MAC: Large firm to hard swelling with nodular or irregular surface. Cut section shows areas of haemorrhage & necrosis and cyst formation.

MIC: There are 5 microscopic types:

1-Mixed (pleomorphic) malignant tumour:

-It arises on top of mixed benign tumours.

2-Mucoepidermoid carcinoma:

-It arises from the duct epithelium.

-It contains both squamous and mucous secreting epithelium.

-It may be of low grade, intermediate or high grade malignancy.

3-Epidermoid (squamous cell) carcinoma.

-It arises from the duct epithelium.

-It is a rare tumour and usually arises in the submandibular gland.

4-Adenocarcinom:

-The cells are arranged in acini.

5-Adenoid cystic carcinoma (cylindroma) :

-The cells are arranged in columns around central cavities giving *Swiss cheese appearance.*

-It is famous for perineural invasion for a long distance and recurrence after many years.

Spread:

1-Direct spread:

-Intrinsic: within the gland infiltrating the facial nerve.

-Extrinsic: infiltrates the surrounding structures (skin, masseter, mandible & oral cavity).

2- Lymphatic: to deep cervical LNs.

3-Blood: To lung & bones.

Complications:

1-Nerve paralysis: facial palsy in parotid tumour and hypoglossal & lingual nerves palsy in submandibular tumours.

2-Cosmetic disfigurement.

3-Infection. & fistula formation.

4- Ulceration. & haemorrhage.

5-Distant metastasis.

Incidence:

They are common over 50 years.

Clinical picture:

1-Pain in the parotid region that may be referred to the ear.

2-Swelling which is: -Single.

-Site: in the parotid region.

- Size: variable (rapidly growing).

-Shape: irregular.

- Surface : nodular or irregular

-Skin over, may be fixed to it

-Surrounding structures:

*Facial nerve, may be infiltrated (facial palsy)

*Masseter muscle may be infiltrated

-Special characters

*It shows the classic characters of parotid swelling (see before)

*The swelling is slightly tender, warm with ill-defined edge.

-The cervical LNs, may be enlarged, hard, painless & may be fixed.

Investigations:

1-Fine needle aspiration cytology (FNAC).

2-Isotope scanning: malignant tumours give cold spot.

3-CT, MRI: To assess operability.

Differential Diagnosis:

From other swellings in the parotid (see before) or submandibular region (see later).

Treatment:

I-Operable cases

A-Parotid tumors:

-Radical total parotidectomy

*Removal of the whole gland sacrificing the facial nerve
*Probably the masseter & part of the mandible (if infiltrated)
*Total block neck dissection.

} as one mass.

N.B. The facial nerve may grafted or replaced by the hypoglossal nerve.

B-Submandibular tumours:

Radical total submandibular sialadenectomy)

*Removal of the whole gland & LNs.
*Propably the adjacent mandible & tongue (Commando)
*Total block neck dissection.

} as one mass.

II-Inoperable cases

1-Palliative resection.

2-Radiotherapy has a minor role.

Prognosis:

It is not favourable.

NB. Parotidectomy for benign tumours is more difficult than that done for malignant tumours because the facial nerve is sacrificed in the latter while it should be saved in the fomer.

The locally malignant tumours

They are tumours that spread mainly by local invasion, very rarely spread by lymphatics or blood

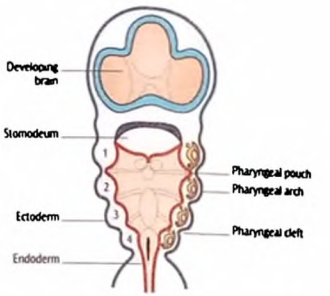
They include:

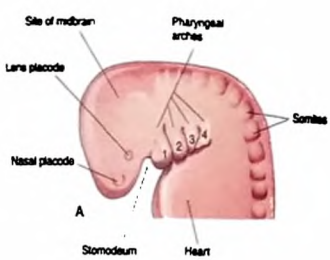
1- Basal cell carcinoma
2-Admantinoma
3- Osteoclastoma
4-Recurrent fibroma of Paget
5- Carcinoid tumour
6- Bronchial adenoma
7- Craniopharyngioma
8- Mixed tumour of the slivary gland
9- Syringoma (of the sweat gland)
10- Tricho-epithelioma

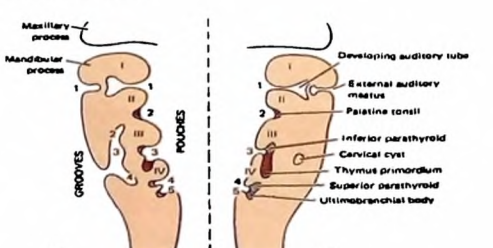
Neck Surgery

Embryology

- The neck is developed from six pharyngeal, visceral or branchial arches but the fifth arch fails to develop in human embryo.
- These arches are circular cylinders that give the neck a wavy appearance.
- They are formed due to proliferation of mesoderm between ectoderm & endoderm.
- These arches are separated by grooves or sulci where the ectoderm & endoderm come in close contact with each other.
- These sulci are termed ***branchial clefts*** from their ectodermal side and termed ***pharyngeal pouches*** from their endodermal side.
- The second arch grows much more rapidly than the other arches so that it forms a shelf infront of the other arches which covers them and descends to fuse with the sixth arch (now it is the fifth)
- Fusion of the second arch with the 6th arch leads to
  - * Loss of the wavy (archy) appearance of the neck (it becomes smooth).
  - * Formation of a pouch (sinus) that is buried under the second arch called the ***cervical sinus*** which normally disappears but it may persist forming *branchial cyst* or the second arch fails to fuse completely with sixth arch and a congenital *branchial fistula* results.

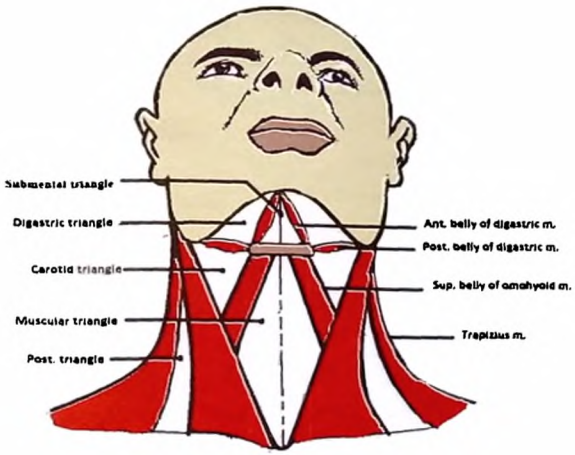






**Recently, embryological studies proved that the branchial system consists of only four arches and the first is the one which overhangs the rest of clefts.*

Anatomy



Triangles of the neck

The neck has seven triangles anteriorly & two posterior triangle:

- **One submental triangle:** between the two anterior bellies of digastric muscle & the body of hyoid bone.
- **Two muscular triangles:** between the anterior midline of the neck, superior belly of omohyoid muscle and the lower part of the anterior border of sternomastoid.
- **Two digastric triangles**: between the anterior & posterior bellies of the digastric muscle & the lower border of the mandible.
- **Two carotid triangles:** between the superior belly of omohyoid muscle, posterior belly of digastric muscle & the upper part of the anterior border of sternomastoid muscle.

The two posterior triangles: between posterior border sternomastoid muscle, anterior border of the trapezius muscle & middle 1/3 of clavicle.

The cervical Lymph Nodes

The cervical lymph nodes (LNs) are classified into:

1- Level I (the submental and the submandibular group)

They are divided into (IA) in the submental triangle and (IB) in the submandibular triangle.

2- Level II (upper jugular group)

The LNs that lie around the upper one third of the internal jugular vein (IJV) from the skull base to the level of C4.

They are divided by the spinal accessory nerve into IIA and IIB

3- Level III (the middle jugular group)

The LNs that lie around the middle one third of the IJV from the level of C4 to the level of C6 (cricoid cartilage)

4- Level IV (the lower jugular group):

The LNs that lie around the lower one third of the IJV from the level of C6 to the level of the clavicle

Levels of Cervical LNs

5- Level V (posterior triangle group)

They are divided by the spinal accessory nerve also into level VA and VB

6- Level VI (the anterior or visceral group)

The LNs that surround the midline viscera (trachea & oesophagus) from the hyoid bone to the superasternal notch.

7- Level VII (the superior mediastinal group)

The LNs present in the upper anterior mediastinum.

Congenital Anomalies

1- Congenital torticollis (sternomastoid tumour) .
2- Branchial cyst .
3- Congenital branchial fistula .
4- Cystic hygroma .
5- Thyroglossal cyst.
6- Cervical rib .
7- Sequestration dermoid cyst.

Congenital torticollis (twisted neck)

(Sternomastoid tumour)

Aetiology:

It is not a congenital anomaly. It is due to birth injury most properly due to infarction of a portion of sternomastoid muscle as a result of injury of the sternomastoid branch of the superior thyroid artery during forceful rotation of the head at birth.

Pathology:

Infarction of a portion of the muscle leads to swelling then fibrosis & shortening of the muscle.

Complications:

Sternomastoid tumour

1-Torticollis : the head tilts to the same side and rotates to the other side.

2-Facial & cranial asymmetry .

3-Development of a new macula on the affected side to keep the intensity of vision horizontal.

Clinical Picture:

-The condition usually presents in the 2nd or the 3rd week of life as oval firm swelling in the middle portion of the muscle *(Sternomastoid Tumour)*. It is a false tumour.

-Later ,the infant develops gradual torticollis.

Treatment:

-Early before torticollis, physiotherapy may prevent the deformity

-Later after torticollis , sternomastoid tenotomy is needed.

2-Branchial Cyst (کیس خیشومی)

Aetiology:

Branchial Cyst

Persistent cervical sinus

Pathology:

Site: In the carotid triangle partially covered by the anterior border of sternomastoid muscle below the angle of the mandible.

Composition:

- The cyst is lined with stratified squamous epithelium surrounded by a fibrous wall.
- The wall is rich in lymphoid tissue that predisposes to infection.
- It contains mucoid fluid rich in cholesterol.
- It is connected to the wall of the pharynx by a fibrous band passing behind the tonsil.

Complications:

1-Infection: with abscess formation.

2-Fistula: when ruptures or surgically drained or incompletely removed.

3-Carcinoma: it may rarely develope branchiogenic carcinoma.

Incidence:

It usually appears in the third decade (20-30 year), more in males.

Clinical picture:

-Slowly growing painless oval cyst with smooth surface in the upper part of the neck (the carotid triangle) behind the anterior border of sternomastoid muscle.

-Contraction of the muscle makes the cyst bulge out as it slips from underneath its ant. border.

-It is opaque in trans-illumination

-It is not attached to the surrounding structures.

Investigations:

Aspiration may differentiate between it and cold abscess by its high cholesterol content.

Treatment:

Complete surgical excision of the cyst with its band up to its end in the wall of the pharynx.

Branchial Fistula

Aetiology:

1-Congenital branchial fistula :

It is due to failure of complete fusion between the second & the fifth branchial arches, so the cervical sinus remains communicating with the exterior.

2-Acquired branchial fistula:

It is due to infection of branchial cyst that ruptures or surgically drained or due to incomplete excision of a branchial cyst.

Cong. branchial Fistula

Pathology:

	Congenital branchial fistula	Acquired branchial fistula
Site	It lies on the lower part of the anterior border of sternomastoid muscle.	It lies on the upper part of the anterior border of sternomastoid .
Aetiology	Congenital .	It occurs on top of a cyst.
Age	It is dated since birth.	It occurs in young adults.

Investigations:

Fistulography: with lipidol

Differential Diagnosis:

From other sinuses & fistulae of the neck (7):

1-Thyroglossal fistula. 2-Sinus of subhyoid bursitis. 3- Salivary fistula.

4-T.B sinus. 5-Actinomycosis sinus.

6- Sinus of osteomyelitis of the mandible.

Treatment:

Complete excision of the fistula from its opening and through its course between the two carotid arteries up to its end in the lateral wall of the pharynx in the *supratonsillar fossa of Rosenmuller* .This may need multiple transverse incisions (stepladder (سلم) incision) in the neck.

-The operation may be complicated with injury to major vessels & nerves.

Neck Swellings

I- Swellings in the midline of the neck

1- Nodule (or cyst) in the thyroid isthmus.

2- Thyroglossal cyst.

3- Dermoid cyst (submental or suprasternal).

4- Lymph nodes (submental , prelaryngeal or pretracheal).

5- Subhyoid bursitis: a sausage shaped transluscent cystic swelling below the hyoid bone.

6- Cold abscess: usually suprasternal.

7- General Swellings:

a) From skin: sebaceous cyst & abscess.

b) From S.C: lipoma.

c) From vessels: haemangioma..

d) Aneurysm of innominate artery.

II – Swellings in the lateral side of the neck

A- Swellings in the carotid triangle

1- Lymph nodes (the commonest).

2- Nodule or cyst in the thyroid lobe (upper pole).

3- Branchial cyst.

4- Laryngocele.

5- Carotid body tumour.

6- Cold abscess.

7- General swellings:

a) From skin: sebaceous cyst , abscess.

b) From SC.: lipoma.

c) From vessels: - aneurysm of the carotid vessels, haemangioma.

d) From lymphatics: lymphangioma.

e) From muscles: sternomastoid tumour & haematoma

B – Swellings in the digastric triangle.

1-Submandibular LN. enlargement.

2-Submanbibular salivary gland enlargement.

3-Swelling of the mandible: tumours , subperiosteal abscess.

4-Plunging ranula.

5-Ludwig's angina.

6-Cold abscess.

7-General swellings as before.

C- Swellings in the posterior triangle.

1-Lymph node enlargement.

2-Cystic hygroma.

3-Prominent cervical rib.

4-Pharyngeal pouch.

5-Pneumatocole.

6-Cold abscess.

7-General swellings: as before

N.B. Pneumatocele: is herniation of the apex of he lung through the suprapleural membrane *(Sibson's fascia)* in emphysamatous patient.

Cervical lymphadenopathy

* Many clinically palpable lymph nodes are normal superficial nodes

*Normal nodes have an elliptical configuration; while pathological nodes, particularly those containing tumour, often become round

*The most common causes of cervical lymph node masses are inflammatory and neoplastic.

Inflammatory cervical lymphadenopathy

Acute cervical lymphadenitis is common (particularly in children) and is due to an infective process in the upper aerodigestive tract.

- The mass is usually painful and can result in an abscess
- The patient is usually toxic and the primary infective process is usually evident.

Tuberculous adenitis may lead to collar –stud abcess

Neoplastic cervical lymphdenopathy

- *Hodgkin's and non-Hodgkin's lymphoma*: painless ,rubbery with systemic symptoms
- *Metastatic lymphadenopathy:* painless ,hard ,fixed with evident primary

The Larynx

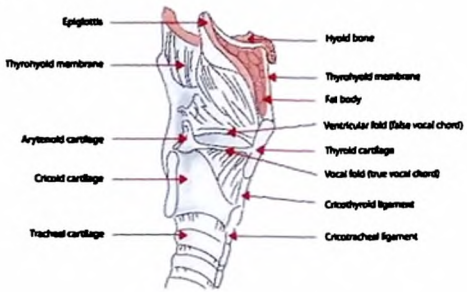
-It is the organ of phonation and constitutes an important part in air passage due to its sphincteric action. It is composed of:

** Three unpaired cartilages*

Thyroid, cricoid & epiglottis

**Three paired cartilages*

Arytenoid, corniculate & cuneiform cartilages.



Sagittal section of the larynx

Muscles of the larynx

I-Extrinsic muscles:

They are the suprahyoid & infrahyoid muscles.

II-Intrinsic muscles:

They are numerous and all are supplied by the recurrent laryngeal nerve except the cricothyroid muscle which is supplied by the external laryngeal nerve.

Laryngocele

Definition:

It is herniation of the mucosa of the larynx through a weak point, which is the opening in the thyrohyoid membrane that transmits the sup. laryngeal artery & internal laryngeal n.

Trumpet players cyst

Aetiology:

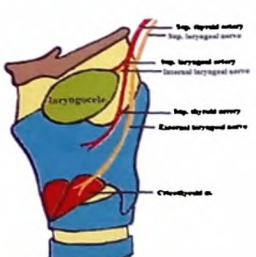
Due to increased intraluminal pressure as in glass blowers & trumpet players → diverticulum.

Clinical picture:

-It is usually unilateral cystic swelling in the carotid Δ.

-It is translucent in trans-illumination.

-It is reducible on compression but reappears when the patient blows his nose with his mouth closed.



Laryngocele

Differential Diagnosis:

From other swellings in the carotid triangle.

Treatment:

Surgical excision

The pharynx

Anatomy

It is a muscular tube about 14 cm in length.

-It begins from the lower surface of the occipital bone.

-It ends at the level of the 6th cervical spine where it is continuous with the oesophagus.

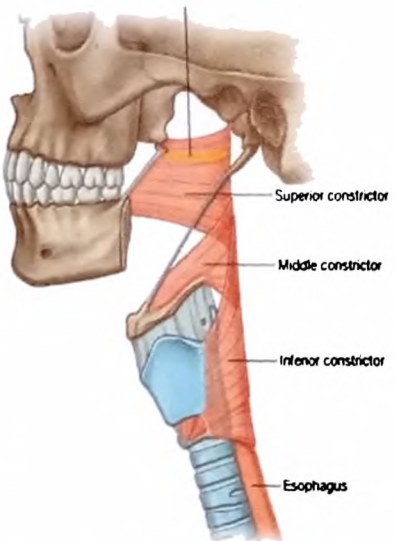
-It is divided into three parts:

1-Nasopharynx: The part above the soft palate.

2-Oropharynx: The middle part between the soft palate & the upper border of the epiglottis.

3- Laryngeopharynx: The part below the upper border of the epiglottis down to the lower border of the cricoid cartilage.

-It has three constrictors ,superior, middle & inferior.



Anatomy The pharynx

N.B. The inferior constrictor muscle is formed of two parts, the thyropharyngeus muscle & the crico-phyaryngeus muscles. The later is considered the distal sphincter of the pharynx.

Pharyngeal diverticulum (pouch)

(Zenker's diverticulum)

Definition:

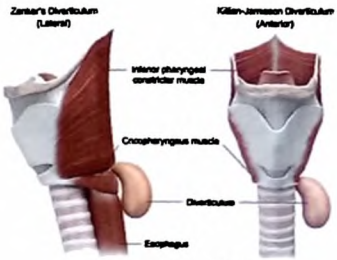
It is herniation of pharyngeal mucosa through *Kilian dehiscence*, which is a weak part in the inf. constrictor of the pharynx between thyro-pharyngeal and crico-pharyngeal parts.

Aetiology:

It is due to muscle incoordination (spasm of cricopharyngus muscle) leading to increased intraluminal pressure and herniation of mucosa.

Pathology:

At first it is globular then enlarges and becomes pear shaped & descends to appear in the posterior triangle of the neck.



Pharyngeal diverticulum

Incidence:

It is more common in old age.

Clinical picture:

-History of long standing dysphagia.

-Regurgitation of foul undigested food that may evoke severe attacks of choking & cough.

-The patient may hear & feel a gurgling sound in the neck with drinking.

-Later, a soft reducible cystic swelling appears in the posterior triangle of the neck especially on the left. side. Pressure over swelling leads to emptying of malodorous fluid in the pharynx with attack of chocking .

-Increasing dysphagia is due to compression of the pouch on the cervical oesophagus.

Investigations:

1-Barium swallow:

It shows the diverticulum.

2-Pharyngeoscopy .

It is risky as it may induce perforation of the pouch.

Barium meal

Treatment:

1-Diverticulectomy.

The ideal treatment is to excise the diverticulum together with crico-pharyngeal myotomy to decrease the intraluminal pressure.

2-Diverticulopexy:

It means invagination of the diverticulum into the lumen and closing the point of invagination (the defect) myotomy may be added.

Endoscopic division of the septum

4-Anastomosis between the diverticulum and the oesophagus: or better endoscopic division of the septum between the pharyngeal pouch & the oesophagus especially in old unfit patient It may be done in multiple stages

Postcricoid carcinoma

Definition:

Carcinoma of the hypopharynx at the level of cricoid cartilage (C6).

Aetiology:

It may occurs on top of *Plummer Vinson syndrome* which is much more common in females

N.B, *Plummer Vinson syndrome* is a rare disease of young females consists of

3As.	Anaemia (iron def.)	Atrophic glossitis.	Achlorohydria
& 3Ss:	Splenomegaly.	Spooning of nails	Spasm of cricopharyngeus

Pathology:

Site: The tumour arises from pharyngeal mucosa covering the back of the cricoid cartilage.

Mac: Usually a fungating mass.

Mic: Squamous cell carcinoma.

Spread: -*Local:* to the surrounding structures.

-*Lymphatic:* to cervical LNS.

-Blood spread to the lung.

Complications:

1-Ulceration: bleeding & infection.

2-Obstructions: -oesophageal → dysphagia -Laryngeal: → stridor & dysnea.

3-Hoarseness of voice. *4-Spread & metastasis.*

Incidence:

It is more common in ♀ (4 : 1). However in Egypt, as Plummer Vinson syndrome is rare the incidence is more common in ♂

Clinical picture:

1-Pain:

-Sense of discomfort or FB. sensation in the pharynx is an early symptom.

-Pain referred to the ear (through Arnold's nerve) that increases with swallowing comes later.

-Dysphagia is the main symptom ,but it may be late in tumours of the pyriform fossa.

2-Swelling(s):

-Neck swellings are due to metastases in cervical LNs. They'r hard, painless and may be fixed.

-It may be the first or the only manifestation especially in tumours of the pyriform fossa.

3-Post-cricoid carcinoma may cause broadening of the larynx with loss of the normal click felt during side to side movement of the larynx.

4-Picture of complications: e.g. Hoarsness of voice, foul odour of the mouth (infection).

Investigations:

1-Plain X-ray. It may show widening of the space between the larynx & vertebrae.

2-Barium swallow: It shows an irregular filling defect in the pharynx.

3-Pharyngeoscope & laryngeoscopy: They show the tumour ,but it may be difficult to see tumours in the pyriform fossa due to marked congestion & lakes of mucin & saliva (froth) over the tumour. Biopsy should be taken.

Treatment:

1-Operable cases:

-They are treated with total laryngopharyngectomy with block dissection of lymph nodes.

-The pharynx is replaced by

1-skin flap from the neck or chest wall.

2-pectoralis major myocutaneous flap.

3-gastric pull up in the neck.

4-colon interposition: It is anastomosed to the floor of the mouth above & the stomach below (antesternal or retrosternal).

-The operation is ended with permanent tracheostomy.

II- Inoperable cases & anaplastic tumours:

They are treated with external irradiation.

Prognosis:

It is poor even after surgery or radiotherapy.

Carotid body tumour

(Potato tumour)

Definition:

It is a low grade malignant tumour arising from the chemo-receptor cells in the carotid body (chemodectoma).

Carotid body tumour

Pathology:

Site: The carotid body is situated in the bifurcation of the common carotid artery, and rarely bilateral.

Mac: oval hard swelling (potato like) with smooth or lobulated surface and variable size.

Mic: It resembles pheochromocytoma.

Spread: It gives lymphatic spread in 20% of cases (late) and distant metastsis in 4% of cases.

Incidence:

Common in middle age.

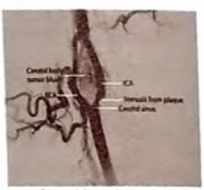
Clinical picture:

- Painless slowly growing swelling in the carotid triangle with smooth or lobulated surface & hard consistency.
- It moves from side to side but not vertically.
- Pulsation of the external carotid artery is felt over the swelling and may be thrill & bruit.
- Pressure over the swelling may cause fainting attacks (carotid sinus syndrome).
- In 20% of the cases, it gives rise to lymph node metastasis.

Investigations:

1-Carotid angiography: It shows

- Wide separation of the two carotid arteries (pathognomonic).
- High vasculrity of the tumour.



Carotid angiography

Differential Diagnosis:

From other swellings in the carotid triangle especially LNs. All swellings are superficial to the external carotid except the carotid body tumour is deep to it.

Treatment:

- Surgical excision: either subadventitial or excision with resection of the carotid arteries and graft replacement. Ligation of internal carotid artery may cause hemiplegia ,so it should be bypassed by a graft before excision of the tumour to avoid interruption of the flow. Some surgeons advise hypothermia during operation to decrease the 0_2 need by the brain.
- Unfit patients may be paliated by irradiation.

Tumours of the head & neck that give rise to pain referred to the ear are:

1- Parotid tumours — pain referred through the auriculotemporal nerve

2- Tongue carcinoma — pain referred through the lingual nerve

3- Postcricoid carcinoma
4- Thyroid carcinoma
5- laryngeal carcinoma

} pain referred through the Arnold's nerve

Neck (block) dissection operations

Definition:

Removal of cervical LNs as one block.

Types:

1-Total block dissection (TND):

Removal of all cervical LNs of the neck.

Indications:

Carcinomas of the head & neck.

We remove:

1-All LNs in one side of the neck on both anterior & posterior triangles of the neck .

2-Sternomastoid muscle & its nerve (spinal root of accessory).

3-Carotid sheath & internal jugular vein.

4-All structures mentioned in suprahyoid block dissection.

We should preserve:

The carotid arteries, vagus, phrenic & hyposglossal nerves .

2- Modified neck dissection (MND):

Total block neck dissection with preservation of one or more of the following structures : spinal accessory nerve, IJV and sternomastoid muscle.

When bilateral total block dissection is indicated we should perform total block dissection on the affected side & preserve the IJV on the other side for adequate venous drainage for brain.

3- Selective neck dissection (SND):

- Removing selected groups (levels) of lymph nodes according to the site and stage of the primary cancer such as

1) Supraomohyoid ND : Removing level I,II & III LNs

2) Antrolateral ND: Removing level I – IV LNs.

3) Lateral ND: Removing level II, III & IV LNs.

4) Postrolateral ND: Removing level II – V LNs

5) Anterior (centeral) ND: Removing level VI LNs

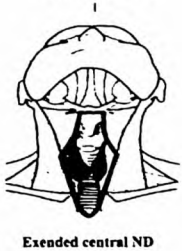
6) Exended central ND: Removing level VI & VII LNs.

Supraomohyoid ND

Antrolateral ND

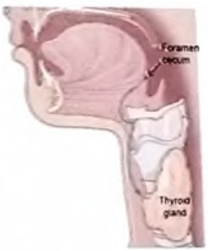
Lateral ND

Postrolateral ND


Exended central ND

THE THYROID GLAND

Embryology



-The thyroid gland develops as an endodermal down growth between the first & the second branchial arches called the thyroglossal duct. It descends from the foramen caecum between the anterior 2/3 and posterior 1/3 of the tongue.

-During its descent it contacts *the ultimobranchial bodies* from the fourth branchial arch which shares in the formation of the lateral lobes of the thyroid and through which, the parafollicular C cells (developed from the neural crest) reach the thyroid.

-When the gland reaches below the cricoid cartilage, it divides into two lobes then the thyroglossal duct obliterates and disappears.

Anatomy

-The thyroid gland is a butterfly shaped endocrine gland 15-25 gm in weight. It consists of two lateral lobes, isthmus and pyramidal lobe .the size of each lobe is (2 x 1 x 1 inch)

-It extends from the middle of the thyroid cartilage (oblique line) to the level of the 6th tracheal ring. The isthmus covers the 2nd, 3rd and 4th tracheal rings.

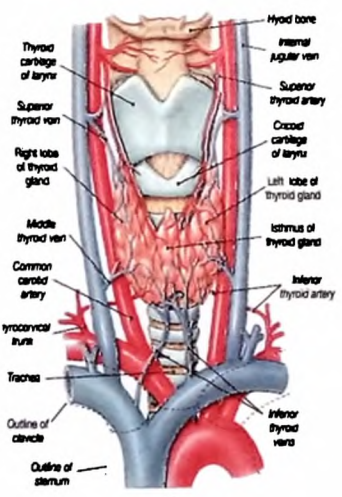
-The thyroid has two capsules:

**True capsule*: from condensation of its CT.

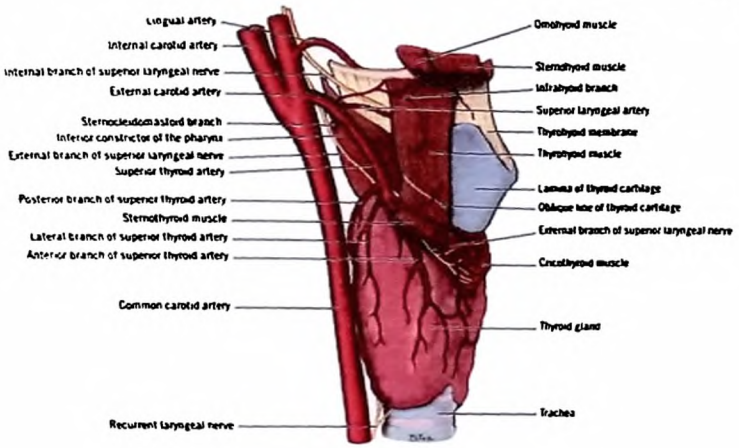
**False capsule*: from the pretracheal fascia.

-The thyroid gland is related:

*Anteriorly: to skin, SC. fat, platysma, deep cervical fascia, pretracheal muscles. Each sternomastoid muscle covers the lower pole of the corresponding lobe.



Anterior Veiw



Lateral Veiw

*Posteriorly: It is related to two tubes (trachea & esophagus), two cartilages (thyroid & cricoid) two muscles (cricothyroid & inferior constrictor of the pharynx.) two nerves (the recurrent laryngeal and external laryngeal nerves).

Blood supply:

Arterial:

- *Inferior thyroid artery*: from the thyrocervical trunk from the first part of the subclavian art.
- *Superior thyroid artery* :from the external carotid artery.
- *Thyroidea ima artery* :from innominate artery or the aortic arch.

Venous:

- *Superior thyroid veins*: to the internal jugular veins.
- *Middle thyroid veins*: to the internal jugular veins.
- *Inferior thyroid veins* :to the brachiocephalic veins.

Lymphatic drainage:

1-Ascending vessels:

- Medially: to the prelaryngeal LNs. -Laterally: to the upper deep cervical L.N.s.

2- Descending vessels:

- Medially: to the pretracheal LNs. -Laterally: to the lymph nodes of recurrent chain.

Applied anatomy

- The pretracheal fascia that encloses the thyroid is thickened and condensed in the posteriomedial aspect of the thyroid gland to form *the suspensory ligament of Berry* which is attached to the cricoid cartilage. So the thyroid gland is attached to the larynx and moves up & down with deglutition.

- The pretracheal fascia is attached to the oblique line of the thyroid cartilage thus preventing the enlarging gland to pass upward.

- The branches of external carotid artery can be represented by the sentence *Some American Ladies Found Our Pyramids So Magnificant*

1-Superior thyroid art. 2- Ascending pharyngeal art. 3- Lingual art. 4- Facial art.
5- Occipital art. 6- Postauricular art. 7- Superficial temporal art. 8- Maxillary art.

- The superior laryngeal nerve (branch. of the vagus) divides into two branches:
  - Internal laryngeal nerve, which with sup. laryngeal art. pierces the thyrohyoid membrane to the larynx. It is sensory to larynx above the level of vocal cords.
  - The External laryngeal nerve, which descends with the sup. thyroid art. It is motor to the cricothyroid muscle. Care should be taken when ligating the sup. Thyroid artery during thyroidectomy not to injure the external laryngeal nerve.

- The recurrent laryngeal nerves (branch. of the vagus) descend downward where:
  -The Lt. one hooks around the aortic arch then ascends.
  -The Rt. one hooks around the Rt. subclavian art. then ascends.
  - Both of them ascend in the trachea-esophageal groove to enter the larynx just behind the *suspensory ligament of Berry* (anatomical landmark for the recurrent laryngeal nerve).
  -They are motor to all intrinsic muscles of the larynx except cricothyroid & sensory to the larynx below the level of vocal cords. Great care should be taken when ligating the inf. thyroid art. during thyroidectomy to preserve the recurrent larngeal nerves

Histology:

The thyroid gland consists of follicles, which are normally lined with cubical epithelium. They form the colloid that is stored in the lumen of the follicles. Adjacent to the follicles, there are the parafollicular C cells that secrete calcitonin.

Physiology:

Synthesis of the thyroid hormones : It passes through the following steps:

1-*Iodide trapping* : the thyroid cells have high affinity to pick up inorganic iodine from blood.

2-*Oxidation of Iodide*: the inorganic iodine is converted into nascent iodine by *peroxidase enz.*

3-*Binding or organification*: Iodination of the tyrosine (inside the thyroglobulin molecule) into mono and di-iodotyrosine by the *iodinase enz.*

4-*Coupling*: coupling of mono-iodotyrosine and di-iodotyrosine to form **T3** and coupling of two molecules of di-iodotyrosine to form **T4.**

5-***Storage***: the thyroglobulin molecules are stored in the lumen of the thyroid follicles in the form of colloid.

6-***Release***: when the hormones are required, the thyroglobulin is taken up by the cells and acted upon by *the protease enzyme* to release free T4 & T3 to the circulation. T3 &T4 are bound to serum protein called thyroid binding globulin (TBG) & thyroid binding prealbumin (TBPA). Only very small part of the hormones are free in the serum with an equilibrium with the protein bound part. The free fraction of the thyroid hormones, is the biological active part.

-T3 is rapid acting (within few hours), while T4 is slow acting (within 4-14 days).
-T3 is more potent (4 times) than T4 but, with shorter half life.
-T_4 is converted into the more active form T_3 in the tissues by a process called peripheral conversion.

The pituitary thyroid axis:

- The synthesis and release of thyroid hormones are controlled by the Thyroid Stimulating Hormone (TSH), which is in turn controlled by the Thyrotropin Releasing Factor (TRF) from the hypothalamus.
- Both TSH and TRF are in a negative feedback relation with the serum level of T_3 & T_4.
- TSH. not only increases synthesis and release of the thyroid hormones but also, increases the size (hyperplasia & hypertrophy) and vascularity of the thyroid gland.

The physiological effect of the thyroid hormones (T_3 & T_4):

1. They stimulate oxygen consumption in all tissue (↑↑ metabolism).
2. They increase glucose utilization by the tissues and glycogenolysis in the liver.
3. They stimulate catabolism of protein.
4. They decrease serum cholesterol.
5. They increase the sensitivity of tissues to catecholamines.

Congenital anomalies

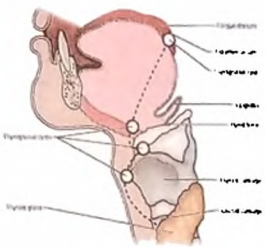
1-Thyroglossal cyst

Aetiology:

It is due to persistent unobliterated portion of the thyroglossal duct (tubulodermoid).

Pathology:

Site: any site in the course of thyroglossal tract from foramen caecum to the thyroid ismuth but the commonest site is below the hyoid bone (infrahyoid).



Thyroglossal cyst

Structure:

- A thin wall cyst lined with stratified columnar, cubical, or squamous epithelium.
- It may contain islets of thyroid tissue and may be connected to lymphatics of the neck which make it liable to infection.
- It contains clear viscid or mucoid fluid.
- It is connected with a thin fibrous band to the hyoid bone.

Complications:

Infection → abscess which drains spontaneously or surgically → fistula.

Incidence:

It may be dated since birth but commonly, it appears in childhood.

Clinical picture:

- A tense cystic swelling in the midline of the neck or slightly to the left usually just below the hyoid bone.
- It moves up & down with deglutition and moves upward with protrusion of the tongue
- The fibrous track connecting it to the hyoid may be palpable.

Investigations:

Thyroid scan to ensure that the thyroid gland is in its place.

Differential diagnosis:-

From other swellings in the midline of the neck.

Treatment: *Sistrunk operation.*

Complete excision of the cyst with its track up to the foramen coecum of the tongue. To ensure complete excision, the central part of the hyoid is resected.

Thyroglossal fistula

Aetiology : always acquired.

1-It follows spontaneous or surgical drainage of an infected thyroglossa

2-It may follow incomplete excision of the cyst with its track.

Clinical picture:

- There is a history of an abscess that was drained spontaneously or surgically followed by a persistant small opening that continues to discharge serous or seropurulent discharge.
- Characteristically,the opening moves up with protrusion of the tongue with formation of cresentic skin fold above.
- It moves up & down with deglutition. Its fibrous track may be palpable.

Pathognomoic sign

Treatment :

Sistrunk's operation.: Ladder steps incision may be required

2- Ectopic thyroid

Definition:

Thyroid tissue congenitally present outside its normal site.

Pathology:

Site: Any site along the course of thyroid descent mostly at the base of the tongue (lingual thyroid).

* Aberrant thyroid: means thyroid tissue congenitally present outside the course of thyroglossal duct. It may lie low in the neck, larynx. or mediastinum.

Lingual thyroid

The term "*lateral aberrant thyroid*" was applied to thyroid tissue that found included in enlarged cervical LNs. but it was found to be metastases from hidden papillary carcinoma of thyroid.

-*Strauma ovarii*: is not an ectopic thyroid but part of ovarian teratoma, which may cause hyper-thyroidism or undergo malignant changes.

Clinical picture: (of lingual thyroid):

-Firm nodule at the base of the tongue may be noticed in infancy.

-It may enlarge with each menstruation causing dysarthria, & dysphagia.

-It is liable to traumatic ulceration & bleeding.

Treatment:

- L- thyroxin is given to reduce its size and avoid complications.
- Excision of the ectopic tissue if it causes complications. if it is the only thyroid tissue in the body, thyroid hormones are given for replacement.

3-Congenital aplasia or hypoplasia : cretinism.

Goitre (الدراق)

Definition: Goitre is an enlarged thyroid gland.

Aetiology & classifications :

Enlarged thyroid gland

I- Metabolic Goitre:

A- *Cretinoid goitre:* (hypofunction goitre).

B- *Simple goitre*: normal function (euthyroid) goitre.

1- Diffuse hyperplastic goitre:

a- Physiological goitre. b- colloid goitre.

2- Simple nodular goitre:

a- Solitary thyroid nodule. b- Multinodular goitre.

C- *Toxic goitre*:(hyperfunction goitre):

1-Diffuse toxic goitre: Grave's, Basedow's , or primary toxic Goitre).

2-Toxic nodular goitre: Plummer's , secondary toxic goitre.

3-Toxic nodule.

II- Inflammatory goitre or thyroiditis:

1-Acute bacterial (pyogenic) thyroiditis.

2- Subacute (de Quervain's , viral) thyroiditis.

3- Auto immune (Hashimoto's) thyroiditis.

4- Riedel's thyroiditis (woody, ligneous thyroid).

III- Neoplastic:

A- Benign: adenomas.

B- Malignant: -Primary: papillary, follicular, anaplastic, medullary, & lymphoma.

-Secondary: metastasis is very rare.

V- Miscellaneous:

Amyloidosis, tuberculosis, & syphilis.

I-Cretinoid Goitre

Aetiology:

Congenital enlargement of the thyroid with congenital deficiency of thyroid hormones due to

1-Congenital enzymatic deficiency.

2- Intake of antithyroid drugs during pregnancy.

Pathology:

Deficiency of thyroid hormones leads to elevated level of TSH, which induces huge enlargement of the thyroid (goitre) together with manifestations of cretinism.

Clinical picture:

I- Manifestations of cretinism:

1- Depressed nasal bridge with puffiness of eye lids.

2- Large lips with large protruding tongue.

3- Spade shaped hand (square with short fingers).

4- Constipation with pot belly abdomen and umbilical hernia

5- Mental retardation and delayed dentitions and milestones.

II- Huge goitre:

Treatment:

1. Thyroid hormones replacement to promote normal growth and development and to reduce the size of the gland.
2. Surgical subtotal thyroidectomy if it causes pressure symptoms.

N.B. Pendred syndrome : a syndrome of 3 **D**s [**D**yshormongenesis (goitre), **D**eafness **D**warfism] due to congenital deficiency of peroxidase enz..

II- Simple Goitre

1-Physiological goitre

Aetiology:

During period of physiological stress e.g. puberty, pregnancy, lactation...etc., there is increased demands for thyroid hormones, so the hypothalamus secretes TRF. which releases TSH from the pituitary that in turn stimulates thyroid to produce more hormones. When there is relative iodine deficiency, there will be marked hyperplasia & hypertrophy of the thyroid (physiological goitre)

Pathology:

The follicles undergo diffuse hyperplasia (increase in number) and hypertrophy (the cells become more tall columnar instead of normal cubical) the colloid becomes markedly reduced.

Complications and fate:

1. *Involution* : In most cases, the goitre subsides and the gland returns to its normal.
2. *Partial involution* : in some cases the gland partially involutes but it returns larger with each cycle. When cycles are repeated, nodular goitre supervenes.
3. *Hyper involution* : in rare occasions → colloid goitre.

Incidence:

It is common in adolescent girls.

Clinical picture:

-Goitre, which has the following characters **5 Ss :**

small, smooth, symmetrical, soft, symptomless

Treatment:

I - Prophylactic: Iodized table salt.

II - Curative: -Reassurance of the patient and her parents.

- L . thyroxin 0.3 → 0.1 mg to suffice the need and decrease the gland size.

2- Colloid Goitre

Aetiology:-

Hyperinvolution of the cell, when the stress is over. The cells seem as they store more colloid for the next stress.

Pathology:

The follicles are distended with abundant colloid and become lined with squamous cells.

Colloid Goitre

Complications:

1-It may turn nodular goitre.

2-Pressure symptoms are rare inspite of its large size due to its softness.

3- Disfigurement.

Incidence:

It is common in females between 15-30 years.

Clinical picture:

Large goitre ,symmetrical, smooth , soft with disfigurment.

Treatment :

-Early: L thyroxin as before.

-late with huge goitre: subtotal thyroidectomy.

3- Nodular Goiter

Aetiology :

Irregular involution of the gland due to aberration of repeated cycles of hyperplasia / involution.

The causes are:

I- Iodine deficiency : which may be

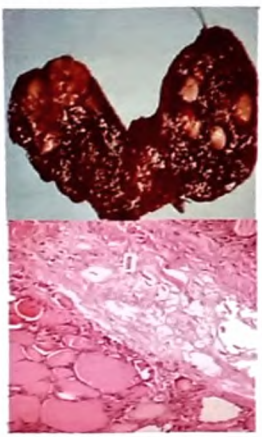
1. *Absolute* : deficient intake in areas where the soil, water and food are poor in Iodine (endemic goitre) as in Oasis & mountains.

2. *Relative* : due to increased demands to thyroxine as in puberty, pregnancy & lactation.

II - Goitrogenic substances:

1- *Diet* : - Cyanogens in cabbage and cauliflower.
- Excess calcium or fluoride.
- Pollution of water with human or animal excreta.

2- *Drugs* :- Antithyroid drugs.
- Sulfur containing drugs e.g. Sulphonamides .
- Iodine containing drugs e.g. cough mixtures .



III- Enzymatic deficiency: within the gland (hereditary).

1- *Peroxidase deficiency* : Pendred syndrome (3 Ds).

2- *Dehalogenase deficiency* .

Pathology:

MAC: -The gland is asymmetrically enlarged with variable consistency. The cut surface shows variable sized nodules with grayish white fibrous tissue inbetween.

MIC: -. The gland shows areas of normal follicles, areas of hyperplastic follicles and areas of hyperinvoluted follicles distended with colloid,. Increased fibrous stroma.

Complications :

1-Secondary thyrotoxicosis: in about 30%

2-Cyst formation: if haemorrhage occurs in it, spasm of pretracheal muscles occurs leading to compression on trachea and suffocation. Urgent thyroidectomy is indicated.

3- Calcification : It leads to formation of hard nodule (DD of malignant tumors).

4-Carcinoma : malignant changes occurs in 5-8 % of cases.

5-Compression manifestations :

a- *Trachea*: in large goitres causing dyspnea. In some longstanding cases, prolonged compression on tracheal cartilaginous rings leads to their softness (tracheomalacia). When the gland is removed, it leaves the trachea unsupported → collapse & suffocation (The goitrous gland supports the trachea by pulling evenly on its walls).

b- *Oesophagus, RLN, and internal jugular vein*: pressure on these structures occurs only in

1- Retrosternal extension 2- hage. in a cyst 3- malignant changes

Incidence:

It is more common in females between 20-50 years (around 35y.)

Clinical picture:

Symptoms:

1- Painless slowly growing swelling in the front of the neck.

2- Dyspnea especially when lying down or during exercise.

3- Symptoms of complications may be the first presentation.

Signs:

- Asymmetrical thyroid enlargement with nodular surface variable size and variable consistency.
- In some cases, only single nodule is felt, but but at operation many satellite nodules may be found around it. It is called " *clinically solitary nodular goitre*".

Investigations:

1-*Thyroid function tests* : to exclude toxic changes.

2-*Thyroid scan:* to check for function distribution, & retrosternal extension.

3- *Indirect laryngoscopy*: to assess vocal cords mobility.

Differential Diagnosis:

Solitary form from other causes of solitary thyroid nodule (STN).

Treatment

Medical treatment is ineffective and surgical removal is recommeded because the changes are irreversible and the goitre is liable to complications.

***I-Solitary form*:**

1-Simple enucleation and resection enucleation are associated with high recurence rate.

2-Hemithyroidectomy includes removal of the diseased lobe + Isthmus + the pyramidal lobe.

The later two are common sites for pathology and recurrence.

II- Multinodular goitre :

1- Subtotal thyroidectomy :

- We leave thyroid tissue (posteriomedial part of each lobe) equal to normal lobe on each side.
- The patient should go on L- thyroxin 0.1 mg / day to avoid recurrence, by suppressing TSH.

2- Total thyroidectomy:

Recent trends are to do total thyroidectomy in all cases of nodular goitre because recuurence is common and injury of RLN and parathyroids is very high in reoperation.

III-Toxic goitre

(Thyrotoxicosis)

Aetiology:

Grave's disease is an auto-immune disease caused by abnormal thyroid stimulating antibodies (Ts Abs) which stimulate the thyroid similar to TSH but with prolonged action (Type V hypersensitivity).

-Ts Abs are members of IgG present in about 80% of patients sera . They are:

-Long Acting Thyroid stimulators (LATS).

- Long Acting Thyroid stimulator protector (LATSP).

- The aetiology of secondary toxic goitre & autonomous toxic nodule is unknown.

Pathology:

There are three pathoclinical types:

I -Diffuse toxic goitre = Primary thyrotoxicosis = Grave's disease

- It occurs in a previously normal gland due to Ts Ab s.

-It occurs in young females usually with exophthalmos.

-The whole gland undergoes hyperplasia & hypertrophy with hyperfunction.

MAC: The gland is symmetrically enlarged with firm fleshy consistency & granular cut suface.

MIC: -The follices are increased in number and lined with tall columnar cells with papillary projections into the lumen which contains scanty colloid.

-The stroma is vascular with A-V shunts & dense lymphocytic infiltration

II - Secondary toxic goitre = *Plummer's disease* .

-It occurs on top of long lasting simple nodular goitre.

-It occurs in middle aged or elderly female.

III - Toxic nodule = autonomous nodule.

-It is not under control of Ts.Abs i.e. It is autonomous.

-It is overactive (Hot nodule on scan) with suppressed surrounding thyroid tissue (due to low TSH secretion).

Microscopic picture

Incidence:

Grave's disease occurs in young adult females while secondary toxic goitre occurs in middle aged or elderly females.

Clinical picture:

1. General manifestations

I-*Metabolic manifestations:*

1-Rapid loss of weight inspite of good appetite.

2-intolerance to hot weather.

3- excessive sweating.

II – Nervous *manifestations:*

1- Insomnia & night mares. 2- Irritability & anxiety.

3-Fine tremors in outstretch hand and protruded tongue. 4-Hyper-reflexia.

III *Cardiovascular manifestations:*

1 Palpitation: tachycardia is always present.

2- Exertional dyspnea.

3-Water hammer pulse due to wide pulse pressure (high systolic + low diastolic Bp.).

4-Arrhythmia :-all types of arrhythmia except heart block.

-Multiple extrasystoles or paroxysmal atrial tachycardia or fibrillation (AF).

5-In late cases, manifestations of heart failure appear.

IV- *Gastrointestinal manifestations:*

1- Increased appetite (polyphagia). 2- Diarrhea.

V- *Urinary manifestations:*

1- Polyuria. 2- Glucosuria.

VI – *Genital (sexual) manifestations :*

1-Early increase of libido but later on, decreased libido and even impotence in men occurs.

2-Early polymenorrhea and hypermenorrhea ,but later, oligomenorrhea or even amenorrhea.

VII *Musculo-skeletal manifestations :*

1-Myopathy : weakness and wasting of proximal muscles of the limbs.

2-Osteoporosis : Depletion of protein from bone matrix leading to generalized bone aches.

VIII *Cutaneous manifestations:*

1- The hand is warm, moist (sweaty). D.D. from psychoneurosis.

2- Falling of hair.

3- Pretibial myxoedema starts as a shiny red plaque of thickened skin over the chin of the tibia. It is due to deposition of mucin like substance under the skin.

IX- *Ophthalmic manifestations* (eye signs):

1- Exophthalmos: (also see clinical)

A- Apparent (mild) exoph.: due to spasm of Muller's muscle leading to widening of the plapebral fissure giving a starry (حملقة) look.

B- True exoph. is due to exophthalmos producing substance (EPS.) that mostly arisies from the anterior pitutary gland in conjunction with TSH. It is classified into:

1- moderate exophth. due to deposition of excess retrobulbar fat.

2- severe exophth. due to additional intraorbital oedema & congestion.

3- malignant exophthalmos due to actual enlargement of the globe with paralysis of extra-ocular muscles.

2- *Other eye signs*:

a- *Stellwag's sign* : infrequent blinking < 4/min. (Normally 5-15/min).

b- *Von Graefe's sign* (lid lagging): the upper eye lid lags in descent behind the eye globe when the patient looks suddenly downward.

c- *Dalrymple's sign* : A rim of sclera is apparent between the upper eye lid & the cornea (normally the upper eye lid covers the upper 1/6 of the cornea).

d- *Joffroy's sign* : lack of corrugation of the forehead on looking to the ceiling.

Mobius sign: Failure of or unsustined convergence on looking to a near object

N.B.- *Muller's muscle* is the deep part of the levator palpebre superioris muscle which is innervated by sympathetic fibres.

-In severe cases (malignant exophthalmos) there are ophthalmoplegia, chemosis of the conjunctiva and corneal ulceration → enophthalmitis.

X- *Reticuloendothelial manifestations*:

There may be mild splenomegaly (10%) and generalized lymphadenopathy.

II- Local manifestations

1- Primary thyrotoxicosis

- The thyroid gland is symmetrically enlarged, firm in consistency with smooth surface.
- It is very vascular ,warm with palpable thrill (light palpation) and machinery murmur can be heard over its upper pole ,as the superior thyroid art. is superficial to the gland.

2- Secondary toxic goitre:

The gland is asymmetrically enlarged with nodular surface.

In some cases (2%) there is no goitre (Masked thyrotoxicosis)

Investigations:

1-Sleeping pulse: counting of pulse during sleep to avoid psychological stress (Normally 70-80/min). It indicates degree of toxicity and used to monitor preoperative control of toxicity by antithyroids: (80-90 b/min → mild, 90-110 b/min → moderate, > 110 b/min → Severe)

2-Hormone assays:

A- Free T3 & T4 : (normally 3-9 & 9-27 pmol /L respectively)

Recently by radioimmunoassay the free part of T3 & T4 can be measured. This is the most accurate test as the free part of the hormones is the biologically active portion.

B- TSH level : (Normally up to 5 MU/L)

It is elevated in hyperthyroidism due to pitutary adenoma and low in other types.

3- Radioactive Iodine studies (Thyroid scan) (^{123}I) or (^{99} Tc)

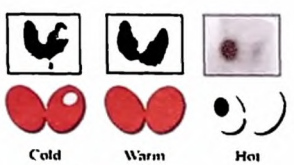
After oral dose of ^{123}I the radioactivity of the gland is screened by gamma camera to detect

1- the size and contour of the gland .

2- the radioactive Iodine uptake.

3- retrosternal extension.

4- the distribution of the tracer to detect:



Thyroid scanning

*Hot nodule = hyperfunctioning = toxic nodule.

*Cold nodule = nonfunctioning → Q malignancy.

*Warm nodule = it doesn't show in scan (It takes ^{123}I as much as the surrounding tissue).

- It can also differentiate between hyperthyroidism in Grave's disease with high uptake and hyperthyroidism in thyroiditis with low uptake.

Precautions before radioactive iodine studies:

1- Drugs containing I_2 e.g. cough mixtures should be discontinued 3 weeks before the test.

2- No contrast radiography should be performed 3 weeks before the test (contrast contains I_2).

3- Radioavtive iodine is given orally (5- 25 microCuries) with milk or fluids so, the patient should be free from GIT troubles.

4- Thyroid auto-antibodies :

a) Ts Ab s(LATS & LATSP) are elevated in Grave's disease in exacerbation.

b) Antimicrosomal & Antithyroglobulin Abs are present in hyperthyroidism due to thyroiditis.

5- Preoperative investigations:

Indirect laryngoscopy (to evaluate the vocal cords mobility), S.Ca^{++} , ECG , Ts.Abs

Differential Diagnosis:

1- from other causes of loss of weight inspite of good appetite

a- D.M. b- Heavy parasitic infestation. c - Malabsorption synd.

2- from psychoneurosis: Anorexia rather than polyphagia is present in psychoneurosis. Also, the hand is sweaty but cold. Sleeping pulse and T3 & T4 are normal.

3- from heart failure, myasthenia gravis, 1ry exophthalmos especially masked thyrotoxicosis

4- from other causes of hyperthyroidism:-

i. Thyroiditis: early cases of de' Quervain's or Hashimoto's thyroiditis may show mild hyperthyroidism due to liberation of hormones from damaged gland.

ii. Thyrotoxicosis factatia : due to intake of thyroxin as a tonic.

iii. Jod – Basedow thyrotoxicosis: thyrotoxicosis due to supplying large doses of iodine to hyperplastic endemic goitre.

iv. Large mass of functioning secondary follicular carcinoma or sturuma overii.

v. Neonatal thyrotoxicosis: Transplacental passage of TsAbs from toxic mother to foetus.

5- Primary toxic goitre should be differentiated from secondary toxic goitre as follows

Items	Primary toxic goitre	secondary toxic goitre
Age	Usually young 20- 30y	Usually old 40-50y
History	Usually–ve (normal gland)	Long lasting nodular goitre
Nervous manifestations	marked	Minimal
Cardiac manifestations	Less marked	Marked
Ophthalmic manifestations	Present	Rare
Thyroid enlargement	Small, symmetrical smooth	Large, asymmetrical, nodular.
Response to ATD.s	better	less
Recurrence after surgery	higher 10-20 %	lower I-2 %

Treatment :-

1- Medical treatment

Indications :

1 -All cases of diffuse toxic goitre.

2- Children & young patients (high recurrence after surgery).

3-Patients refuse surgery or unfit for surgery.

4- Progressive exophthalmos :medical ttt is given till exophthalmos is stable for 6 months.

5- As a preoperative preparation.

Aim :To control toxic *symptoms* and maintain the patient euthyroid till a natural remission.

Lines:

1-Physical & mental rest : tranquilizers & sedatives may be used

2-Inderal : (beta blocker)

- Action: - It controls cardiac manifestations
  - It blocks peripheral conversion of T4 into T3
- Dose: 10-40 mg tds

3-Antithyroid drugs (ATDs) ,They include:

A- K perchlorate & thyiocynates (not used nowadays)

- They block Iodine trapping (step1) i.e. they make the gland hungery.
- They may precipitate thyroid storm if iodine is supplied or they are stopped.

B-Thiourea derivatives ,

Methyl thiouracil, Propyl thiouracil, Methimazole & the most commonly used carbimazole

- Action: 1- They block oxidation, Iodination & coupling (step 2,3,4).
  2- They may have immunosuppressive action on Ts Ab.
  3- Propyl thiouracil ,additionally, blocks the peripheral conversion

  Their effect appears after 7-14 days as they don't affect the already formed T3 & T4.
- *Dose* :- Start with carbimazole 15 mg /8h till euthyroidism , then reduce gradually to 5 mg /8 h for 12-18 months.
- *Side effects* :

1-Allergic reaction.

2-Bone marrow depression (aplastic anaemia & agranulocytosis). The patient is warned that if s/he develops sore throat to stop the drug at once & have a complete blood picture, if agranulocytosis is proved, the patient should receive :

- IV penicillin. - fresh bl. transfusion. -Vit. B complex.

3- Increased size & vascularity of the goitre due to rise of TSH. Persistent tachycardia may develop inspite of treatment due to opening of intraglandular A-V shunts.

4- Cretinoid goitre: if they are taken during pregnancy (foetal thyroid begins to trap iodine at 12th week) or during lactation (ATDs are excreted in milk)

5- Hypothyroidism with overdosage.

- *Contraindications:*

1- Lactation:.

2-Retrosternal or intrathoracic goiters (↑↑ TSH → ↑↑ size → ↑↑ pressure manifestations).

C- *Lugol's Iodine* (5 % Iodine + 10% KI in water):

- *Action*:1- It blocks the release of thyroid hormones (it inhibits protease enz.) i.e. It constipates the gland (step 6).Also, it inhibits organification (steps 3).

2- It blocks the stimulant effect of TSH on the gland so, it decreases the vascularity.

*Its action starts within 24h & maximum effect is within 10-15 days.Its effect declines afterwards (tolerance).It cannot be used for long term therapy and used only in:

- Preoperative preparation to decrease vascularity & toxicity.
- Treatment of thyroid crises.

- *Dose:* start with 5 drops tds then increase gradually to 15 drops t.d.s.
- *Side effects*:

1- Parotid enlargement (salivation) 2-allergy 3- fibrosis around the thyroid.

NB. In pregnancy, ATDs are given in the lowest dose that control toxic symptoms but if they are continued after labour, lactation is prohibited

II – Surgical Treatment

- Indications:

1- Nodular toxic goitre & solitary toxic nodule.

2- Failure of medical treatment or recurrence after successful ttt.

3- Huge diffuse toxic goitre with pressure symptoms.

4- Retrosternal & intra thoracic toxic goitre.

5- Suspicion of malignancy.

- Preoperative preparations:

1- Preoperative investigations as before.

2- Sedatives.

3- Antithyroid drugs (unless contraindicated e.g, retosternal goitre.).

4- Inderal that is continued for l week after operation.

5- Lugol's Iodine 2 weeks before operation (not more) together with ATDs.

* Some surgeons advocate to replace the antithyroid drugs 15 days before the operation with Lugol's Iodine to reduce the size and vascularity of the gland, others ommited Lugol's Iodine.

- Operation:

Subtotal thyroidectomy where we leave part of the gland that is sufficient to maintain the patient euthyroid. This is estimated to be 1/3 of a normal thyroid lobe on each side (about 5-10 gm of the gland) or 1/8 of the goitrous gland.

(Steps, technique & complications see later).

III – Radio-Iodine therapy

- Indications:

1- Patients unfit for surgery (due to age or disease)

2- advanced thyrocardia .

3- Patient refuse surgery & unfit for medical ttt .

4- Recurrent toxicity after surgery.

5- toxic solitary nodule.

All patients should be over 25 years.

- Mode of action :

When radioactive Iodine ^{131}I is trapped by the thyroid gland, it emits *Beta Rays* that destroy the gland. Beta rays have a low penetrability so, they don't damage the surrounding tissues. Its effect appears after 8-12 weeks (ATDs are given concomitantly).

- Dose :

160 μ curies/ 1 gm thyroid tissue (about 5-10 millicuries) once orally. If there is still toxic symptoms, another smaller dose is given 3 months later.

- Disadvantages and complications:

1- The proper dose is difficult to be calculated especially in operated cases.

2- Delayed onset of action.

3- If taken during pregnancy, it may cause foetal anomalies & cretinoid goitre.

4- Myxoedema (up to 80% after 10 years) or recurrent toxicity (improper doses).

5- Risk of inducing thyroid carcinoma after a latent period of about 20 years".

- Contraindications:

1- Young age < 25 years: (risk of malignancy)

2- pregnancy : Teratogenicity.

3- lactation, excreted in milk.

4- Iodine allergy.

5-. secondary toxic goitre with marked nodularity

Treatment of progressive exophthalmos

I-Medical:

1- protection of the eyes from light and dust with dark glasses

2- Salt restriction + Diuretics.

3- Corticosteroids.

4-T_3 ,03- 0.4 mg /day together with ATDs to inhibit EPS.

II – Surgical:

1-lateral tarsorraphy

2-orbital decompression operation (Naffziger's operation).

Retrosternal goitre

Definition

It is not a separate pathological type of goitre. It is merely an anatomical entity.

(Retrosernal goitre = goitre behind the sternum)

Aetiology

1- Retrosternal extension of large goitres (common) helped by gravity & pushing of pretracheal muscles.

2- Goitre in intrathoracic ectopic thyroid (rare)

Pathology

It may be simple, toxic or malignant.

Complications

Pressure complications e.g. dyspnea, dysphagia, hoarseness of voice.

Incidence

It is more common in males due to

1- Short neck. 2- Strong pretracheal muscle.

Clinical picture

Symptoms : those of mediastinal (thoracic inlet) syndrome.

Dyspnea (trachea), dysphagia (esophagus), oedema & congestion of the face (innominate vein),& hoarseness of voice (RLN.).

Signs:

1- Inspection :-

-Engorged neck veins & dilated veins over the sternum.

-Tilting the head to one side or raising the arms till they touch the ear for somewhile cause flushing of the face & dyspnea *(Pemberton's sign)*.

2- *Palpation* :

- The lower border of the cervical goitre is not well defined.
- In ectopic intrathoracic type, the goitre may appear only during swallowing (plunging goitre).

3- *Percussion* :-

Dullness over the manibrum sterni may be elicited.

Investigations :

1- Plain X ray chest , reveals mediastinal mass deviates tracheal air to one side.

2- C.T & M.R.I.

3- Thyroid scan : It can differentiate RSG. From other mediastinal masses.

4- Thyroid function tests.

Differential diagnosis:

1- From heart failure.

2- Form other causes of mediastinal syndrome.

Treatment: (Surgical removal)

1- *For the common type associated with cervical goitre :*

The retrosternal extension is enucleated form the sup. mediastinum assisted by gentle traction of the thyroid lobe. No risk of bleeding as this extension has its bl. supply form inf. thyr. art.

2- *For the rare intrathoracic type :*

Sternotomy is needed as the goitre is deep and has intrathoracic blood supply.

N.B: For toxic retrosternal goitre antithyroid drugs are contraindicated as they cause enlargement of the goitre → ↑↑ pressure manifestations.

Thyroiditis

1- Acute bacterial thyroiditis

Aetiology --It is a rare condition, which may follow pharyngeal or oral infection or blood born infection.

Pathology - diffuse inflammation of the thyroid gland. Abscess formation is rare.

Clinical picture -Fever and other constitutional manifestaions.

- Pain and tenderness over the gland.
- Marked dysphagia and limitation of neck movement.

Investigations - Bl. picture: marked leucocytosis.

Treatment by antibiotics and drainage of abscess if collection is proved

2- Subacute viral thyroiditis

(De Quervain's thyroiditis)

Aetiology - It is caused by virus (influenza or mumps).

Pathology Characterized by granuloma formation with giant cells.

Incidence: It affects middle age females.

Clinical picture: -Painful. gland, fever, malaise, & firm irregular goitre.

-In some acute cases, there are toxic symptoms.

Treatment: There is dramatic response to predinsolone 10-20 mg / day for one week then gradual withdrawal for one month.

3- Autoimmune thyroiditis

Hashimoto's disease = Lymphadenoid goitre

Aetiology: - It is an autoimmune disease due to abnormal formation of antibodies against thyroglobulin (sequestrated antigen).

Pathology: The gland is moderately enlarged, nontender ,and firm rubbery in consistency.

Incidence: It affects middle-aged females especially at menopause.

Clinical picture: -The onset is usually insidious with only goitre.

-Early cases may show mild toxic symptoms and late cases show myxoedema.

Investigations: - detection of antithyroglobulin antibodies in the serum.

- Thyroid function tests and thyroid scan according to stage of disease.

Treatment: - L. thyroxin with corticosteroids give good response.

-Surgery is indicated in marked pressure symptoms or suspicion of malignancy.

4-Riedel's thyroiditis

= Ligneous (woody) thyroiditis

Aetiology: It was thought to be a chemical inflammation due to leakage of colloid outside the follicles but, it is most probably a collagen disease.

Pathology: There is an extensive fibrosis infiltrating the gland and surrounding structures (muscles, trachea, oesophagus and carotid sheath).

Incidence: It is a rare disease affecting young adults of both sexes.

Clinical picture: -The gland is mildly enlarged but stony hard (woody thyroid).

-Pressure symptoms are very marked.

Treatment: Isthmectomy with biopsy is indicated to relieve pressure and differentiate it from anaplastic carcinoma.

-Thyroidectomy is impossible & radiotherapy is ineffective due to marked fibrosis.

Tumours of the thyroid

I- Benign tumors

A-Follicular adenomas:

1-*Embryonal adenoma:* the cells are arranged in columns or sheets.

2-*Foetal (micro follicular) adenoma:* The cells are arranged in minute follicles.

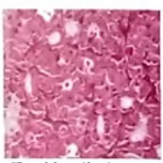
3-*Simple adenoma :* The cells are arranged in moderate sized follicles.

4-*Colloid (macrofollicular) adenoma :* The cells are arranged in large follicles.

5-*Hurthle cell adenoma :* the cells are large containing granular acidophilic cytoplasm.

B - Papillary adenomas:

Cystic spaces with papillary projection .It is considered malignant.



Hurthle cell adenoma

Complications:

1- Cystic degeneration.

2- Haemorrhage into a cyst.

3- *Malignant transformation* (5-10%).

Clinical picture:

Usually presents with solitary thyroid nodule.It is firm ,& well defined nodule in the thyroid.

Investigations:

See solitary thyroid nodule.

Differential diagnosis:

From other types of solitary thyroid nodule especially involutional nodule:

True adenoma	Involutional nodule
- Rare.	- Common.
-Actually solitary.	- Not solitary.
-Different in structure from other gland.	- Similar in structure to surrounding.
-Well encapsulated	- Poorly capsulated.
- It compresses the adjacent tissue	- It does not compress the adjacent tissue.

Treatment:

Hemithyroidectomy with biopsy.

NB. Thyroid paradox phenomenon: Most of the thyroid cysts are so tense that they are felt as solid masses and some of thyroid solid masses are so soft that gives them pseudo-fluctuation and they are felt as if they are cystic

II- Carcinoma of the thyroid

Aetiology:

I Predisposing factors:

1- *Irradiation to the neck* :(irradiation to T.B . lymph node or radioactive iodine).

2- *Genetic predisposition* :some cases of medullary carcinoma are familial.

3- *Prolonged TSH stimulation*: as in endemic areas with low I_2 intake.

II- Precancerous conditions

1-Nodular goitre: (5-8 % risk).

2-*Hashimoto thyroiditis*: may be complicated by papillary carcinoma or malignant lymphoma.

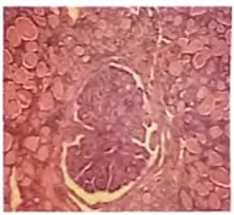
3-Thyroid adenomas:(5-10 % risk).

Pathology:

There are five main pathological types:

I- Differentiated Thyroid carcinoma (DTC): It includes:

1-Papillary carcinoma 80%



Papillary carcinoma

- Common in children & young adults.
- *MAC*:- localized soft (slowly growing) nodule. Cut section shows cystic areas with papillary projections.
- *MIC*: large cystic areas with intracystic branching papillae covered with malignant cells. *Psammoma bodies* (few scattered calcifications) may be present.
- *Spread* : - mainly by lymphatics: cervical LNs. mets falsely named lateral aberrant thyroid.

2- Follicular carcinoma (10%):

- Common in middle age.
- *MAC*: small irregular ill-defined mass (usually pulsating). Cut section gives gritty sensation. Grayish areas with hage. & necrosis are present.
- *MIC*: The malignant cells tend to arrange themselves in complete or incomplete acini or follicles (well differentiated)or in clumps (poorly differentiated).
- *Spread*: mainly by blood spread.

3- Hurthle cell carcinoma

- It is regarded as a more aggressive variant of follicular cacinoma
- It has earlier lymphatic spread and lower response to RAI and hence worse prognosis.

4- Mixed tumours:

- Various proportions of the above types are present in the tumour

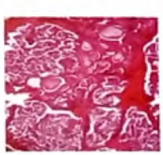
- *Character:* DTC is characterized by the following
  - It may be multicentric or multifocal (especially the papillary type).
  - It concentrates radioactive I_2 (RAI) and may be functioning(especially the follicular type).
  - it is hormone (TSH) dependent(especially the papillary type).

II- Anaplastic (undifferentiated) carcinoma (5%):

- It is common in old age.
- *MAC*: - large hard (rapidly growing) irregular infiltrating mass. Cut section show marked local infiltration with areas of hage & necrosis.
- *MIC*:- Spheroidal cell carcinoma. The cells are form clumps separated by fibrous stroma.
- *Character :* -gives a temporary response to radiotherapy.
  -Its spread is mainly local beside lymphatic and blood spread.

4-Medullary carcinoma (5%):

- It arises from parafollicular C cells
- Non-familial type: affects old age above 50y.
- Familial type (autosomal dominant character): affects young age. The mutant gene (RET gene) is located on chromosome no.10. It may be in the form of Multiple Endocrine Neoplasia (*MEN*) syndrome (see below) or familial non-*(MEN)* medullary carcinoma.
- *MAC:* hard nodular mass.
- *MIC*: sheets and cords of malignant cells in hyaline amyloid stroma.
- *Spread:* by both lymphatic and blood spread.
- *Character:* It secrets calcitonin (used as a tumour marker), and may be prostaglandin or serotonin (5 HT) causing diarrhea.



Medullary carcinoma

5- Lymphoma (non-Hodgkin lymphoma) rare:

- It usually affects old age patients with long standing Hashimoto thyroiditis.
- *MAC & MIC* like lymphoma elsewhere in the body.
- *Character:* It gives good response to radiotherapy & chemotherapy.

Clinical picture:

Although each type has its own characters, the following features are suggestive of malignancy:

1-Rapidly growing goiter or recent rapid enlargement of longstanding goitre..

NB. Very rapid enlargement with pain denotes hage rather than malignancy

2-Pain which may be referred to the ear through *Arnold's nerve* (auricular branch of the vagus)

3-Pressure symptoms: dyspnea (trachea), dysphagia (esophagus) or change of voice (RLN).

4-Hard, irregular, fixed thyroid swelling with high vascularity.

5-Impalpable carotid pulsation (*Berry's sign*). The carotid is encased with the tumour.

6-Metastasis in the cervical LNs may be the first presentation (dormant cancer).

7-Distant metastases may be detected (they are sometimes pulsating especially in the scalp).

Investigations:

Scalp mets from follicular carcinoma

1- Radioiodine scanning: it is helpful:
- *cold nodule* is suspicious of malignancy.
- extent of local & lymphatic spread.

2- Thyroid U/S: It differentiates solid from cystic swelling.

3- Tumour markers: - S. thyroglobulin.
- S. calcitonin in medullary carcinoma.

4- Biopsy: from the thyroid mass or from enlarged cervical LNs. Biopsy is taken either by:
- Fine Needle Aspiration Cytology (FNAC): It can't differentiate between follicular adenoma and carcinoma as the main histological difference between them is the capsular and vascular invasion which don't appear in FNAC.
- Trucut needle biopsy is risky (hage., RLN & other important structures injury).
- frozen section biopsy during surgery.

5- Chest CT & bone scan to detect metastases.

6- Indirect laryngoscopy, to check for RLN involvement.

Differential diagnosis:

Malignant nodule should be differentiated form other causes of STN.

Treatment:

I- <u>Differentiated thyroid carcinoma :</u>

A- Surgery:

- Total thyroidectomy is the most accepted procedure because:
  - the tumour is multicentric.
  - Total thyroidectomy removes all thyroid tissue that may compete with the tumour for RAI in scanning or RAI therapy.
- Central and selective neck dissection should be added if LNs are palpable.
  We should preserve:
  - at least one parathyroid gland.
  - RL.N. on both sides unless infiltrated.

NB. Central neck dissection entails removal of the prelaryngeal, pretracheal and paratracheal LNs may be with the thymic tongue.

B- RAI therapy:

- Anti thyroid drugs may be given preliminary to stimulate TSH. to increase the tumour uptake of RAI.
- Radio-Iodine therapy in a big doses (100-200 millicuries) is given to destroy the residual tumour cells & metastases.

C- Hormonal therapy:

- Post RAI therapy L. thyroxine in big doses to suppress TSH and tumour growth.

II- Anaplastic carcinoma:

- It is almost always inoperable because of rapid local spread.
- Radiotherapy (external radiation not radio-iodine as it does not concentrate Iodine).
- Surgery is indicated only in very early tumour without metastases or late cases with marked dyspnea for isthmectomy. try to avoid tracheostomy.

III- Medullary carcinoma:

-Total thyroidectomy + central and selective block neck dissection.

- In MEN II cases, pheochromocytoma is operated first as it is more life threatening.

If genetic studies show the mutant gene, prophylactic total thyroidectomy should be done at the age of 5 year for MEN IIA and 1 year for MEN IIB.

IV- Malignant lymphoma:

Treated in the same lines for anaplastic carcinoma.

Prognosis:

Several prognositic scoring systems have been developed. The most popular is the AGES system (Age, Grade, Extent & Size of the tumour). The AMES system replaces Grade with Metastasis.

- The papillary carcinoma (the most favorable) has 10-year survival of about 95 %.
- The follicular and medullary carcinomas have 10 year survival of about 80 %.
- Anaplastic carcinoma has the worse prognosis; 1 year survival is less than 10 %.
- Lymphoma has a good prognosis except if it is part of widespread lymphoma.

The parafollicular C cells which are developed from the neural crest constitute part of the Amine Precursor Uptake and Decarboxylation (APUD cells) that may share other APUD cells in the body in Apudoma syndromes or Multiple Endocrinal Adenomatosis (MEA)

MEA I = ***Werner's syndrome***: consists of adenomata of the pituitary, parathyroid, thyroid, adrenal cortex and pancreas.

MEA II = ***Sipple syndrome***: consists of medullary carcinoma of the thyroid + parathyroid adenoma + Pheochromocytoma

It may be associated with mucosal neuromas of the tongue & lips (MEA IIB syndrome).

Solitary thyroid nodule

Definition:

STN. is a clinically single thyroid nodule in an otherwise impalpable gland.

Aetiology & Types:

1- Solitary nodular goitre (metabolic nodule).

2- Solitary toxic goitre.

3- True thyroid adenoma (follicular) 30%.

4- Malignant nodule (1ry & rare 2ry) 10-20% .

5- Rare causes (tuberculosis & syphilitic gumma).

Incidence:

About 3% of adults in UK. & USA have S.T.N. Three to four times more common in ♀.

Diagnosis

I-Clinically:

1-Solitary nodule in extremes of ages (specially in ♂) is suspicious of CA.

2- Manifestations suggesting malignancy (mention) denote malignant nodule

3-Toxic manifestations in toxic nodule.

4-Large percent of cases are asymptomatic.

II – Investigations:

1-Thyroid function tests.

2 Thyroid scan:

Cold nodule. → in malignant nodule or cyst.

Hot nodule. → in toxic nodule

Warm nodule → in metabolic nodule or functioning adenoma.

3-Thyroid ultrasound: It differentiates between cystic & solid nodules.

4- Biopsy: (to exclude malignancy):

- Fine needle Aspiration cytology (FNAC).

-Trucut needle biopsy is risky (hage., RLN & other important structures injury).

-Excisional biopsy including frozen section biopsy.

Treatment: According to pathology.

1-Hemithyroidectomy: removal of the affected lobe + isthmus +biopsy is indicated in metabolic nodule, toxic nodule and follicular adenoma.

2-Total thyroidectomy: in malignant nodule + appropriate therapy.

Subtotal thyroidectomy

Principle:

To remove most of the diseased gland and leave part of the gland, that maintains euthyroid state:

1- In toxic goitre, we leave tissue about 1/3 of a normal lobe or 1/8 of hugely enlarged gland.

2- In simple multinodular goitre, we leave tissue equal to normal lobe in each side.

Pre-operative preparation:

see before.

Operation:

Anaesthesia: General with endotracheal intubation.

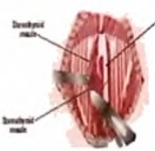
Position: supine with extended neck.

Incision: Kocher's collar incision

- 1 inch above the suprasternal notch a transverse incision extending from the posterior border of sternomastoid muscle in one side to the posterior border of the other muscle. It cuts through skin & platysma.

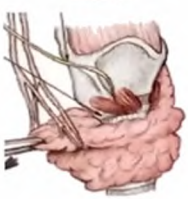
Steps:

1- *Dissection of the skin flaps*: the upper flap is dissected to the upper border of the thyroid cartilage & the lower to the manubrium.



2- *Exposure of the gland:* the infrahyoid muscles with the deep fascia are incised in the midline then they are either retracted laterally or cut transversely near their upper pole to avoid complete denervation of the muscle (ansa cervicalis supplies them from their lower pole). We cut muscles in:

1) huge goitre. 2) toxic goitre. 3) malignant goitre.



3- Devascularization of the gland :

- The middle thyroid vein is ligated first to facilitate Mobilization of the lobe and avoid its rupture → air embolism.

4- *Resection of most of the gland* :

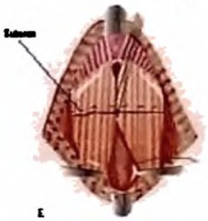
By slanting incision to preserve the posteriomedial part of the gland

a- to maintain the patient euthyroid.

b- to avoid injury of recurrent laryngeal or parathyroid gland.

5- *Haemostasis* :

It is secured by suturing the capsule of the remaining part of the gland to the pretracheal fascia.



6- *Closure*: in layers with one drain in each side.

Complications:

1-Hemorrhage : It may be

a- Primary hage. : during operation.

b- Reactionary hage: within 24 h. : - It is usually due to slipping of sup. thyroid art. ligature. If not sufficiently drained, the blood will accumulate beneath the infrahyoid muscles and press on trachea → dyspnea (serious).

-The wound should be opened in bed to relieve dyspnea and stridor then transport the patient to the theatre to deal with the bleeder.

c- Secondary hage: 7-10 days after operation due to infection that erodes blood vessels.

2-Injury to the recurrent laryngeal nerve:

The nerve is injured during ligation of inf. thyroid art.

(a) *Unilateral injury:*

a-*Incomplete* : The affected cord is adducted leading to dyspnea on exertion due to narrowing of the glottis.

b-*Complete*: The affected cord lies in the cadaveric position leading to hoarseness of voice that usually improves in few weeks due to compensation of the other cord that crosses the midline to meet its fellow but the patient usually complains of voice fatigue at the end of the day.

Incomplete **Complete**

Unilateral nerve injury

(b) *Bilateral injury:*

a-*Incomplete* : (most serious) both cords will be adducted in the midline leading to stridor & suffocation and needs urgent tracheostomy.

b-*Complete injury* : both cords will lie in the cadaveric position leading to aphonia .

Incomplete **Complete**

Bilateral nerve injury

(3) Injury to the external laryngeal nerve:

Paralysis of the cricothyroid muscle leads to limitation of the force of projection of voice & lowering of the pitch. This usually improves during the first 3 months.

(4) Thyrotoxic crises:

Definition:- a state of acute hyperthyroidism with serious toxic manifestations due to rapid flushing of the circulation with thyroid hormones.

Clinical picture : 5 Hs

1. Hyperthermia with marked sweating.
2- Hypertension both systolic & diastolic with wide pulse pressure.
3- Heart rate markedly rises > 150 beat / minute may be with arrhythmia (AF).
4- Heart failure : may develop (high output failure) with marked dyspnea .
5- Hyperexcitability up to convulsion .

Treatment: Urgent.

1-Ice packs or immersion in cold water bath.
2-Morphia.
3-O_2 inhalation.

4-Indral : I.V. drip under E.C.G. control .
5-Propylthiouracil 100 mg rectally (inhibits the peripheral conversion).
6-Lugol's Iodine 3cc in 1000 cc glucose solution .

7-Digoxin for AF.
8-Diuretics for H.F.
9. Antibiotics to prevent chest infection.

(5) Hypoparathyroidism = tetany

Aetiology: -

1. Accidental injury or removal of the parathyroids.
2. Ischaemia of the gland due to ligation of inf. thyroid art. (Common).

Clinical picture:

-Manifestations appear within 5 days postoperatively in about 5% of cases. It varies in severity:

*Mild cases: tingling & numbness in fingers & toes
(tests for tetany are +ve)

**Moderate cases*: typical carpopedal spasm.

*Severe cases: Laryngeal spasm → stridor (rare).

Carpal spasm

-Most cases are temporary & improve spontaneously but it may be permanent.*Treatment:*

- During attack: I.V.Ca gluconate 10 cc. 10% very slowly.
- In between attacks:↑ Ca intake, vit. D_2 and dihydrotachysterol (AT10).

6- Hypothyroidism (Myxoedema):

It is due to excessive resection of the gland without replacement therapy.

7- Recurrence of hyperthyroidism:

It is due to inadequate resection of thyroid tissue or subsequent hyperplasia of the remnant (young age with high TsAb.s).

8- Malignant exophthalmos:

It is due to drop of thyroid hormones → rise of EPS.

9-Trachomalacia:

In long standing huge goitre.

10-Rare complications:

1- *Surgical emphysema.*

2- Keloid formation specially in low incision near to sternum.

Parathyroid glands

Embryology

The superior parathyroid glands develop from the 4th branchial pouch, while the inferior parathyroid glands develop from the 3rd branchial pouch.

Anatomy

They are four small endocrine glands each is about 2 X 4 X 6 mm.

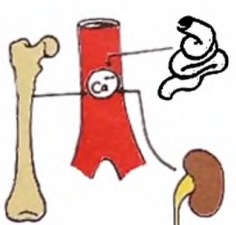
They are yellowish brown in colour and arranged in two pairs.

Superior parathyroid glands: they are more constant in position close to posteriomedial aspect of the thyroid.

Inferior parathyroid glands: they are usually related to the termination of the inferior thyroid artery but they are usually variable in position:

- 40% are at the lower pole of the thyroid.
- 40 % are within the thymic tongue in mediastinum.
- 20% are in rare locations as carotid sheath or post. mediastinum.

Physiology



The parathyroid glands secrete the parathormone hormone which is calcium rising hormone because it:

- Increases resorption of Ca^{++} form bone.
- Increases absorption of Ca^{++} from the gut.
- Increases reabsorption of Ca^{++} from renal tubules.
- Decreases reabsorption of PO_4^- from the renal tubules.

Hyperparathyroidism

Aetiology & Types:

1-Primary hyperparathyroidism: The commonest variety. It is due to adenoma (80%), hyperplasia ,or carcinoma.

2-Secondary hyperparathyroidism: Secondary parathyroid hyperplasia due to hypocalcaemic states e.g. ch. renal failure, & malabsorption syndrome

3-Tertiary hyperparathyroidism: with prolonged stimulation, the reactive hyperplasia acquires autonomy. It secretes excess hormone without stimulation.

Pathophysiology

I-Decalcification of bone

Demineralization of bone leads to deceased bone density with fragility and easy fracture.cysts may appear in bones and hence the name ostitis fibrosa cystica or *Von Recklinghausen's disease*. This cyst is filled with brownish material and hence the name *brown tumour* (false tumour)

Von Recklinghausen's disease

II-Increase in the serum calcium: It causes

1-neuromuscular depression: decreased excitability and conductivity of nervous and muscular (smooth ,skeletal,and cardiac) tissue

2-ectopic deposition of Ca^{++} in tissues (metastatic calcification)

3-increase Ca^{++} excretion in urine causing formation of urinary stones

4-activation of certain hormone e.g gasterin and certain enzymes e.g trypsinogen

Incidence:

It is more common in young adult females (Male: female = 1:3)

Brown tumour

Clinical picture :

I-Nervous manifestations

1-Mental depression and fatigue

3-Restlessness, irritability, personality changes and even neurosis may occur.

II-Cardiovascular manifestations

1-Bradycardia with short Q-T interval

2- Hypertension

Nephrocalcinosis

III-Renal manifestations

1-Polyuria and polydypsia

2-Renal stones (multiple and recurrent)

3-Nephrocalcinosis

IV-Gastrointestinal manifestations

1-Anorexia ,nausea, vomiting and constipation

2-Dyspepsia and may be peptic ulceration

3- Acute pancreatitis may occur.

Kyphosis

V-Musculoskeletal manifestations

1- Muscular weakness with hypotonia and hyporeflexia

2-Generalized bone pain e g backache

3-Deformities e.g kyphosis

4- Pathological fractures e.g vertebral collapse, fracture lower limb during walking. Notablly, The fractures heal rapidly due to hypercalcemia

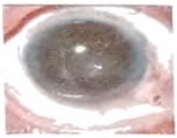
5- Jaw swellings (cysts) that may give egg shell crackling sensation.

Jaw brown tumour

VI-Eye manifestations

1-calcification of the conjunctiva

2-band keratopathy: calcification of the lateral edge of the cornea



Band keratopathy

Investigations:

I-Investigations for hyperparathyroidism:

A- Laboratory invesigations:

1- Serum Ca^{++} & PO_4 : $\uparrow Ca^{++}$,$\downarrow PO_4$

2- Urinary Ca^{++} & PO_4 : $\uparrow Ca^{++}$,$\uparrow PO_4$

3- Radio immune assay for parathormone: ↑ PTH.

B- Radiological investigations:

1- X rayof skull: It appears with multiple erosion giving *pepper pot appearance*

pepper pot appearance

2- X rayof the mandible may show multiple cysts

3- X rayof vertebra show marked softening of bone with indentation of the vertebral bodies by the nucleus pulposus of the intervertebral disc giving Cod Fish spine appearance

Jaw cysts

II-Investigation for localization of parathyroid glands:

1- Ultrasonic scan of the neck. (very helpful)

2- C.T scanof the neck:

3- Thallium. Technetium subtraction scan :

The Idea of this test is that Tcm. is taken only by thyroid gland while Thallium 201 is taken by both thyroid & parathyroid gland. By computerized subtraction of the two captured images, the parathyroids appear as hot spots.

4-Invasive tests' as selective angiography & selective venous sampling by femoral catheter are not in common use.

Cod Fish spine

Differential Diagnosis:

Form other causes of hypercalcaemia:

I-Endocrinal Diseases: (45%) e.g. hyperthyroidism, Addison's disease, & pheochromocytoma.

II-Malignancy: (45%) e.g. metastatic breast carcinoma, paramalignant syndrome, multiple myeloma, & leukaemia.

III-Other conditions (10 %)

1-Increased intake: milk alkali syndrome, & vit D toxicity.

2-Other diseases: Paget's disease of bone, sarcoidosis, & T.B.

3-Prolonged immobilization.

Treatment:

I- Parathyroid adenoma

Resection of the adenoma.

II- Parathyroid hyperplasia

Resect the four glands and implant part of one in sternomastoid muscle to facilitate its re-exploration if needed.

False tumours

They swellings that simulate tumours but they are not neoplasm. They include

1- **Sternomastoid tumour**

2- **Olive tumour** in congenital hypertrophic pyloric stenosis.

3- **brown tumour** of the mandible in hyperparathyroidism.

4- **Pott's puffy tumour** in extradural abscess.

5- **Cock's peculiar tumour** in infected sebaceous cyst.

Chapter VIII

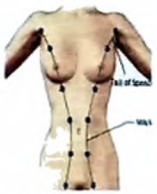
Breast Surgery

The Breast

Embryology

-The breasts develop from two ectodermal ridges called *the milk streaks* extending from the anterior axillary fold to the groin.

-Normally they disappear completely except infront of the chest where 15-20 solid columns of cells arise on each side then they canalize to form the milk ducts.



Milk lines

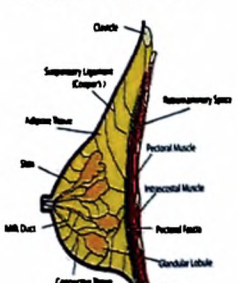
Anatomy

The breast is considered a modified sebaceous gland, because it lies in the superficial fascia.

Histologically:

- It is composed of 15-20 lobes that open independently (by a separate opening in the summit of the nipple through a lactiferous duct (15-20). Each lobe is composed of a number of lobules, which are aggregations of acini.
- The breast gland is embedded in the subcutaneous fat and anchored to the overlying skin and the underlying pectoral fascia by fibrous bands called *ligaments of Cooper* which together with the fat are responsible for the roundness of the breast.

Lobular architecture, extensions and floor of the breast



Ligaments of Cooper

Extensions

Adult female breast extends from the level of the 2nd rib to the level of the 6th rib and from the lateral border of the sternum to the midaxillary line.

Floor:

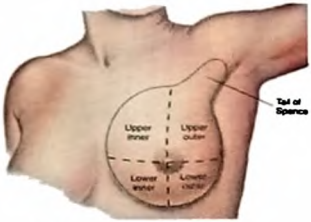
The breast rests on:

-Pectoralis major muscle, (its upper medial 2/3)

-Serratus anterior muscle & ext. oblique muscle (its lower lat. 1/3)

Axillary tail of spence:

- It is a prolongation from the upper outer quadrant of the gland along the lower border of the pectoralis major muscle. Then it passes through an opening in the axillary fascia called *(foramen of Langer)* to enter the axilla.
- It is in close relation to the pectoral group of LNs.
- It is the only portion of the breast that lies deep to the deep fascia.



Axillary tail of Spence

Blood Supply

- Arterial supply:

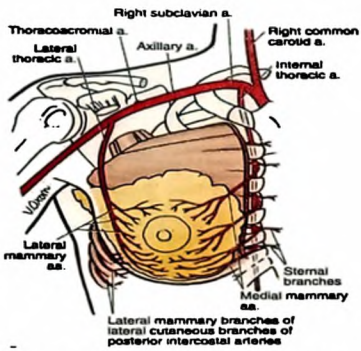
1- axillary artery through the superior thoracic, acromiothoracic & the lateral thoracic arteries

2-internal mammary artery: through its anterior perforating branches.

3- The 2nd, 3rd, 4th intercostal arteries, through their lateral cutaneous branches.

- Venous drainage:

The veins of the breast end in the axillary & internal mammary veins.



Arterial supply of the breast

Lymphatic Drainage:

I-The classical description

There are two sets of lymphatics:

A-Lymphatics of the parenchyma: arranged in two plexuses

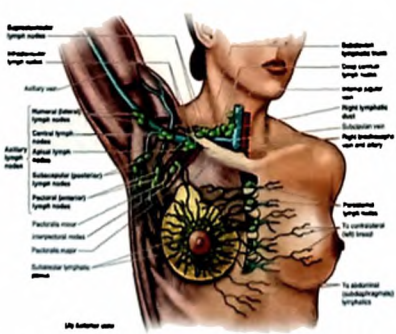
i)-Subareolar plexus of Sappey:

-It drains the superficial part of the gland with the areola & nipple

-It drains into the deep plexus

ii)-The deep plexus: (over the pectoral fascia)

-It drains the deep part of the gland & the subareolar plexus.



Lymphatic drainage of the breast

-It drains into axillary LNs. (pectoral group) and internal mammary LNs .through the pectoralis major muscle.

- Lymphatics passing to the pectoral group will pass to the central group of axillary lymph nodes (at the base of the axilla) then to apical group of axillary LNs (at the apex of axilla) then to the supraclavicular LNs.
- Lymphatic from the inner lower quadrant of the breast communicate with the subperitoneal lymph plexus through the linea alba, so malignant cells of the breast may reach the peritoneal cavity and implant on the liver. And through the falcifrom ligament, malignant nodule may appear around the umbilicus (*Sister Joseph nodule*) .Also ,cancer cells may drop in the pelvis and implant on the ovaries causing (*Krukenberg's tumour*) or nodules in Douglas pouch.

B- Lymphatics of the overlying skin (except nipple & areola):

a- From the outer part to the axillary LNs.

b- From the upper part to the supraclavicular L.N.s (directly).

c- From the inner part to the internal mammary L.N.s. Also they communicate with cutaneous lymphatics of the other breast. Unilateral cancer may give metastases to the other breast through this route if ipsilateral lymphatics were blocked. (*Doubted*).

The Modern description:

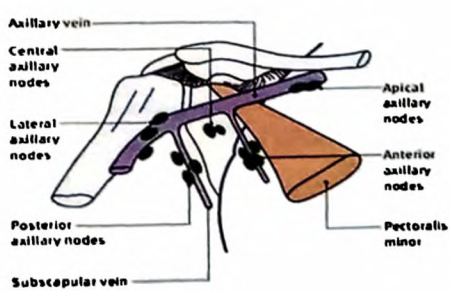
It claims that the role of lymphatic plexuses have minor role and lymphatics accompany the major bl.vessel to drain into:

1- Axillary lymph nodes (75%).

2- Internal mammary nodes (20%).

3- Post. intercostal lymph nodes (5%) near the heads of ribs.

Axillary Lymph Nodes



They are arranged in the following groups:

Group	Site
1-Ant. (pectoral) group.	Deep to the ant. axillary fold (pectoralis minor m.)
2- Post. (subscapular) group.	Deep to the post. axillary fold (Subscapularis m.)
3-Lat. (humoral) group.	Medial to the upper part of the humorous.
4-Interpectoral (Rotor) group.	Between the two pectoralis muscles.
5-Central group.	at the base of axilla.
6-Apical group.	at the apex of axilla.

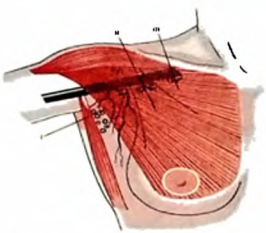
Operative classification of axillary L.N.s:

They are classified into three levels:

1-level 1: - L.N.s below the pectoralis minor muscle.

2-level 2: - L.N.s behind the pectoralis minor muscle.

3-level 3: - L.N.s above the pectoralis minor muscle.



Operative leveling of the axilla

Histology

The ductolobular system is composed of a dual layer of epithelia resting on a basement membrane ("basal lamina") and enveloped by stroma

Physiology:

Hormonal control:

-The duct system is under control of oestrogen, growth hormone & corticosteroids.

-The alveolar (acinar) system is under control of progesterone and prolactin.

Histology of the breast

Congenital Anomalies

1-Amazia: aplasia of one or both breasts.

2-Polymazia: accessory breast along the milk line most commonly in the axilla.

3-Athelia: congenital absence of the nipple.

4-Polythelia: accessory nipple along the milk line.

5-Diffuse hypertrophy of the breast: It needs reduction mammoplasty.

6-Micromazia: It needs augmentation mammoplasty.

Accessory breast

Congenital retraction of the nipple: *(old retraction of the nipple)*

-It appears at puberty, & bilateral in 25% of cases.

-There is no groove around the retracted nipple.

-Suckling is difficult.

-Treatment by: - frequent digital drawing of nipple for 3 weeks.

- application of warmed test tubes.

- *Ashford* operation (purse string of the areola).It is risky.

N.B In recent retraction of the nipple, there is a groove around the nipple and the retraction is always unilateral. It occurs in malignancy & inflammation.

Traumatic Conditions

1-Haematoma

-History of trauma, but it may be negative.

-Calcified haematoma produces hard mass. (D.D carcinoma).

2-Traumatic fat necrosis

Aetiology

-Direct trauma: needle biopsy, & sustained pressure.

-Indirect trauma (sudden contraction of pectoralis major).

Pathology

Trauma leads to hydrolysis of fat with liberation of fatty acids which combine with Ca^{++} of local tissue fluid forming Ca soap which induces foreign body reaction with aggregation of phagocytes and fibroblasts leading to formation of hard mass with white chalky cut surface.

Incidence:

Common in middle aged women.

Clinical Picture:

-History of trauma only in 50% of cases.

-Painless hard lump with retraction of nipple & skin dimpling. *D.D*: cancer.

Treatment: Excisional biopsy.

3-Cracked nipple

Aetiology:

1- Bad hygiene during lactation.

2- Trauma by the baby teeth.

Clinical Picture:

-The nipple is fissured & very painful.

-If neglected it may lead to acute mastitis.

Treatment:

-Stop lactation from the diseased side for 48 hours while evacuating it with a breast pump.

-Wash the nipple and apply antiseptic cream.

-When soreness disappears, resume lactation gradually.

4-Milk fistula

Aetiology:

1- Incision or injury of milk duct.

2- Rupture of subareolar breast abscess.

Treatment:

1-Stop lactation and give Bromocryptin (Parlodel).

2-Fistulectomy: the fistula is removed with the related sector of the gland.

5-Traumatic mastitis

Aetiology:

Ill fitted brassiere in females or ill-fitted suspender in male.

Treatment: Correction of the cause.

6- Open wounds

Like open wounds elsewhere in the body, but during repair, attention should be paid not to injure much milk ducts, to close the dead spaces and to preserve the cosmetic appearance.

Inflammatory Conditions

A-Acute mastitis

1-Neonatal mastitis

Aetiology:

Withdrawal of maternal oestrogen from the baby after birth stimulates the newborn pituitary to produce prolactin that stimulates the baby's breast leading to engorgement & secretion.

Clinical Picture

The breasts are swollen & on the 3rd or 4th day, few drops of milk fluid (witch's milk) can be expressed from the breast. It disappears on the 3rd week.

Treatment:

- If there is no infection, only reassurance of the parents.
- If infection occurs, aspiration of the abscess (may be repeated)
- Incision of the abscess is not advised as it may injure breast bud with no growth at puberty

2-Mastitis of puberty (pubertal mastitis)

Aetiology

Hormonal imbalance during puberty.

Clinical picture:

-It is more common in males during puberty.

-The breast is swollen, tender and indurated. It usually begins unilateral.

-It never suppurates and resolves spontaneously within weeks.

Treatment:

Nothing, but in longstanding cases (for years), local mastectomy is done.

3-Mastitis of mumps

It is usually unilateral and commoner in females.

4-Subareolar mastitis

It is infected *sebaceous gland of Montgomery* or infected hair follicle (furuncle) in the areola.

Clinical Picture:

-Early tender subareolar induration of pea size.

-Later, it may rupture leaving chronic sinus.

Treatment: Early drainage.

5-Mastitis due to milk engorgement

Aetiology:

Obstruction of milk duct by epithelial debris commonly in early lactation & during weaning.

Clinical Picture:

General manifestations: fever due to absorption of milk proteins.

Local manifestations: a sector of the breast becomes swollen, tender & indurated. If neglected, breast abscess develops.

Treatment:

As in pre-suppurative stage in breast abscess.

6- Acute lactational mastitis = Acute breast abscess

Aetiology:

-Causative organisms:

1-Staph aureus (commonest) 2-streptococci (uncommon).

-Mode of infection:

1- Direct (common) through milk duct or fissured nipple.

2- Blood borne (rare).

-Predisposing factors:

1-Milk engorgement, cracked nipple, or bad hygiene of the nipple.

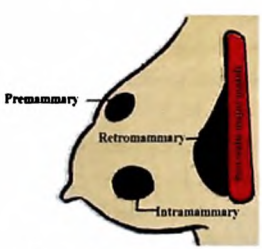
2-Cleansing the baby's mouth with a swab leads to excoriation of its epithelium and infection with staph. which is transmitted to nipple.

Pathology:

It passes into two stages:

-Cellulitis (pre-suppurative) stage.

-Abscess (suppurative) stage.



Sites of breast abscess

Sites:

1- Premammary or supramammary.

2- Intramammary.

3- Postmammary or retromammary.

Complications:

1- Damage to the breast tissue.

2- Toxemia, bacteraemia, pyaemia & septicemia.

3- Spontaneous rupture → chronic sinus.

4- Antibioma or chronic breast abscess (antibiotics in the suppurative stage without drainage).

Incidence:

It is more common in lactating females, especially in early lactation or during weaning.

Clinical Picture:

1-Stage of cellulites:

-General: Constitutional manifestations e.g. fever, headache, anorexia & malaise.

-Local: Manifestations of inflammation e.g. pain, tenderness, redness, hotness & swelling, with enlarged tender axillary lymph nodes.

2-Stage of abscess (localization):

-Fever becomes hectic. -Fluctuation is late.

-Pain becomes throbbing. - Pointing is very late

-Oedema becomes pitting. - Aspiration is positive

Breast abscess

Differential Diagnosis:

From mastitis carcinomatosa (see later).

Treatment:

Pre-suppurative stage→ conservative measures:

1- Antibiotics: penicillinase resistant antibiotics e.g. cloxacillin.

2- Analgesic antipyretics.

3- Hot fomentations.

4-Stop suckling from the diseased side and evacuate it with a breast pump or stop lactation (wean the baby) and suppress secretion with bromocryptine (parlodel) 2.5 mg tab TDS or cabergoline (Dostinex) 0.5 mg ½ tab twice weakly.

Suppurative stage → Drainage. (*Don't wait for fluctuation*).

Role: wide dependent incision under general anaesthesia.

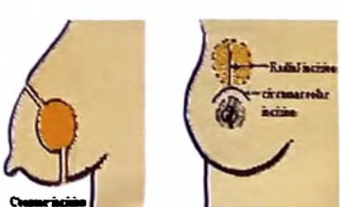
1-Premammary abscess:

Incision where it points (no special direction).

2-Intramammary abscess:

Types of incision:

- Dependent & *Radial incision*: to avoid injury of many milk ducts.
- *Circumareolar incision*: in the skin only then radial in the breast tissue (more cosmetic).
- *Counter incision*: it is more dependent.



Types of incisions

3-Postmammary abscess:

It is drained through an incision in the submammary fold and search for rib osteomylitis, empyema necessitans or TB infection.

B-Chronic mastitis

1-Chronic breast abscess (Antibioma)

Aetiology:

Acute bacterial mastitis with improper treatment due to:

1-Antibiotic therapy alone (without drainage) in the suppurative stage. The pus becomes sterile and surrounded by dense fibrosis (antibioma).

2-Bad drainage: independent incision or incomplete opening of loculi.

Clinical Picture:

-History of acute mastitis usually with antibiotic therapy.

-Breast lump: hard (fibrosis), non-tender mass except in exacerbation.

-Skin dimpling due to fibrosis.

-Nipple retraction.

-Peu d'orange appearance due to obstruction of cutaneous lymphatics by the fibrosis.

-Enlarged tender axillary LNs.

-There may be recurrent episodes of acute exacerbations.

Chronic abscess

Differential diagnosis from carcinoma by:

1-Past history of acute mastitis.

2-Paget's test may be positive.

3-The posterior surface is rounded (not flat as in carcinoma).

4-The axillary L.Ns are firm and tender (not hard & painless).

Treatment:

Excision &biopsy → Differentiation from carcinoma is difficult.
→ the wall is rigid & the cavity persists after simple drainage.

2-Tuberculosis of the breast

Aetiology:

Usually due to retrograde lymphatic spread from cervical, axillary or mediastinal TB. L.N.s.

Clinical Picture

-Multiple abscesses with multiple sinuses discharging caseous material.

-Retro-mammary abscess.

Treatment

-Anti TB. drugs for long period. Rarely mastectomy is needed.

3-Syphilis of the breast: *rare*

A-Primary: chancre in the areola & nipple from diseased partner.

N.B. syphilitic ($) mother is immune against reinfection from her $ baby (Colle's law).

B-Secondary: condylomata lata in the submammary folds because it is a wet surface

C-Tertiary: Gumma

4-Actinomycosis of the breast: - very rare.

It forms sinuses discharging sulfur granules .TTT → mastectomy

5-Mammary duct ectasia: (*plasma* cell mastitis).

Aetiology

Subacute inflammation of the duct system → duct obstruction → distension of the duct (duct ectatsia) with debris & fatty material.

Pathology:

Periductal inflammation with lymphocytes & plasma cells (plasma cell mastitis).

Incidence

-It is common in middle age women.

Clinical Picture:

-Stage of acute inflammation with pain & fever. It usually passes unnoticed.

-Subareolar hard mass with skin dimpling & nipple retraction.

-Creamy green paste like discharge per nipple.

Differential Diagnosis

From other hard masses in the breast especially carcinoma.

Treatment

Excisional biopsy is always needed (difficult differentiation from cancer)

6-Mondor's Disease

Definition: It is thrombophlebitis of the superficial veins of the breast.

Aetiology: unknown.

Clinical Picture

Subcutaneous thrombophlebitic cord of varying length.

Differential Diagnosis

From lymphatic permeation of an occult carcinoma.

Treatment

Restriction of arm movement → spontaneous resolution.

Mammary Dysplasia

= ANDI = Fibrocystic Disease = Fibroadenosis

=Involutional cystic hyperplasia =Ch. interstitial mastitis

Definition:

It is the commonest disease of the female breast due to Aberration of Normal Development and Involution (ANDI).

Bloodgood cyst

Aetiology:

Aberration of normal cycles of development (evolution) and involution due to hormonal imbalance in the form of excess oestrogen and low progesterone.

Pathology:

Site: -It is commonly bilateral but may be unilateral.

- It commonly affects the whole breast but may be localized to one sector.

MAC: -The affected areas become thickened, tough & rubbery in consistency.

-The cut section is white or yellow (never gray) and shows cystic areas sometimes with dominant large one (*Blue domed cyst of Bloodgood*).

MIC: 1-*Adenosis*: increased number of acini & ductules.

2-*Epitheliosis*: increase in epith of acini & ductules→ multiple rows.

3- Papillomatosis: Papillomatous projections inside the ducts.

4-*Fibrosis*: Fibrous tissue replaces fat & elastic tissue.

5-*Cysts formation*:

-Large cysts: are retention cysts due to obstruction of ducts by fibrosis or epitheliosis.

-Small cysts: are degeneration cysts of acini.

6-*Round cell infiltration*: Lymphocytes & plasma cells infiltrate the interstitial tissue.

MIC

Complications:

1-Mastalgia.

2-Cyst formation.

3-Carcinoma (controversial) : mammary dysplasia & carcinoma coincide in 3% of cases. Some consider that epitheliosis is precancerous, while others consider it is just association.

Incidence:

-At any age in the reproductive period. Never before puberty or after menopause.

-Common in unmarried, nullipara or non-lactating apprehensive female.

Clinical Picture:

Symptoms:

1-Pain:

Character: dull aching. Intensity: mild to severe.

Site: breast. Radiation: localized or shoots to shoulders.

Increased: before menstruation. Decreased: during pregnancy & lactation.

2-Lump or better described lumpy breast usually painful.

3-Discharge per nipple: Serous (yellowish), greenish or dark brown.

Signs

-The disease may be unilateral or bilateral, diffuse or localized.

1-Breast:

-Fine & coarse nodules are better felt with the tips of the fingers than with their palmer surfaces, they are tender & mobile.

- Discharge per nipple on milking the breast.

2- Axillary lymph nodes: They may be enlarged, tender & firm (never hard) due to chemical irritation by hypersecretion from hyperplastic acini.

Investigations:

1-*Mammogram.*

2-*Biopsy*: Excisional, incisional or true cut needle biopsy.

Differential Diagnosis:

From other causes of breast lump or discharge per nipple.

Treatment:

1-Conservative

1- Reassurance, tranquilizers, & sedatives.

2- Support the breast with a suitable brassiere.

3- Drugs:

- Bromocryptin (anti prolactin) or Danazol & Tamoxifen (antioestrogen).
- Rarely testosterone is used before menopause (virlizing effect).
- Analgesics to relieve pain.

2-Surgical: Is indicated only in:

1-Localized form with suspicious of malignancy → excisional biopsy.

2-Multiple cysts involving most of the breast
3-Severe resistant intolerable pain.
} Subcutaneous mastectomy with or without augmentation mammoplasty.

IV-Tumours of the Breast

I-Benign tumours

1-Epithelial: Duct papilloma.

2-Connective tissue: Neurofibroma, & lipoma.

3-Mixed: Fibroadenoma, & papillary cystadenoma

1-Duct papilloma

Origin:

Columnar cells lining major lactiferous ducts.

Pathology:

Site: main lactiferous duct near the nipple.

Mac: small soft with finger like projections and a long thin pedicle.

Mic: core of vascular C.T covered with cubical or columnar epithelium.

Complications:

1-*Bleeding per nipple*: due to kink of the pedicle.

2-*Retention cyst*: due to obstruction of the duct.

3-*Malignant transformation*: → duct carcinoma.

Duct papilloma

Incidence:

Common between 35 & 50 years.

Clinical picture:

1-Bleeding per nipple

2-Retroareolar mass (retention cyst).

3-Differential pressure will squeeze bl from the affected duct.

Investigations:

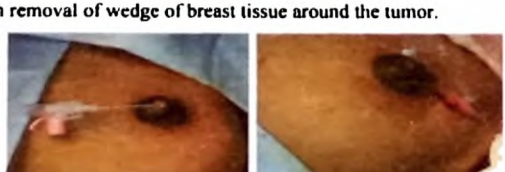
Galactography (ductography or contrast mammography).

Injecting dye in the offending duct shows filling defect.

Treatment:

1- Single → microdochectomy with removal of wedge of breast tissue around the tumor.

2- Multiple→ cone excision of major ducts or simple mastectomy.



Microdochectomy

2-Fibroadenoma

Origin: mixed tumour of epith and C.T. origin.

There are two pathological types:

	Hard (pericanalicular)	Soft (intracanalicular)
Pathology	Mac. Small, slowly growing. Firm swelling. Cut section bulges on cutting. Mic. The ducts are patent and surrounded by dense C.T.	Mac Large, rapidly growing soft swelling. Cut section may shows cytic arears Mic The loose C.T. impinges the ducts and transforms them into slits
Complications	- Rarely turns malignant	- Cystosarcoma phylloids. - Sarcomatous changes.
Incidence	15-35y	30-50y
C/P	- Small, painless, well defined firm mass highly mobile within the breast. (mouse of the breast)	- Large painless well-defined soft mass rapidly growing and may reach, huge size.
Investigation	Mammography & biopsy	Mammography & biopsy
Treatment	Excisional biopsy	Small → excisional biopsy. Large → subcutaneous mastectomy with augmentation mammoplasty

Hard fibroadenoma

Cystosarcoma phylloids

(Serocystic disease of Brodie)

Definition:

It is a giant fibroadenoma showing malignant behavior.

Cystosarcoma phylloids

Pathology:

MAC: - Huge rapidly growing mass with bosselated surface that may ulcerate through pressure necrosis of the overlying skin (not infiltration).

- Cut section: shows cystic cavities into which branching projections of the tumour appear like leaves (phylloids).

MIC: It resembles intracanalicular fibroadenoma but with cystic degeneration and sarcomatous like stroma.

Spread: Malignant tumours spread by blood stream but not by lymphatics.

Incidence:

-It is common above age of 40 years.

Clinical picture:

-Huge mass involving the whole breast with variable consistency and bosselated surface.

-The overlying skin is stretched with dilated veins and may show ulceration but a probe can be put between the tumour and the skin edge (not infiltrated).

Treatment

Simple mastectomy

3-Papillary cystadenoma

Pathology:

- Multiple cystic spaces with many papillary projections.
- It has a great malignant potential.

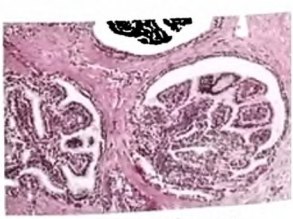
Clinical picture:

-Bleeding per nipple.

-Firm mass (due to papillary projections.)

Treatment:

Local excision and biopsy.



Papillary cystadenoma

Carcinoma of the breast

Aetiology:

I-Predisposing Factors:

1- Genetic Factors:

a- It is commoner in females with positive family history.

b- Milk factor (*Bittner's factor*) is suspected to be oncogenic virus present in the mother's milk that may induce breast carcinoma in the offsprings (only in mice).

c- There are two genes which were identified *BRCA I* (on chromosome 17) and *BRCA II* (on chromosome 13). They are incriminated in causation of a hereditary form of breast CA.

2- Geographical Factors:

It is commoner in western countries and USA. and uncommon in Asia and Japan. This may be due to genetic or dietary factors.

3- Endocrinal (Hormonal) factors:

a- Cancer breast is common in breasts that deny their functions i.e. It is commoner in spinster, nullipara & non lactating females.

b- More in females with early menarche or late menopause (prolonged exposure to oestrogen).

4- Exposure to Radiation.

a- Nuclear war.

b- Medical purposes (diagnostic or therapeutic).

5- Dietary Factors:

a- Diet rich in saturated fatty acids predisposes to cancer.

b- Cancer breast is commoner in obese women.

6- Drugs.

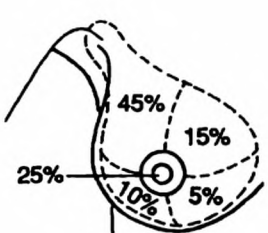
1-Breast carcinoma is common in females taking contraceptive pills and males under oestrogen therapy (e.g. for cancer prostate).

2- Prolonged treatment with reserpine.

II-Premalignant conditions:

1-Duct papilloma:→ duct carcinoma.

2-Mammary Dysplasia: may be precancerous.



Site incidence

Pathology:

Site: It is commoner in the upper outer quadrant because it comprises most of the breast gland including the axillary tail of Spence (see the figure).

(WHO Classification)

I-Non invasive (non infiltrating) Carcinoma or carcinoma in situ.(5%)

1-Intraductal carcinoma:

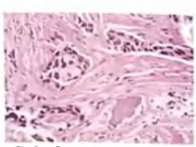
The malignant cells didn't invade the basement membrane.

2-Intra-acinar carcinoma (lobular carcinoma in situ)

II-Invasive Carcinomas

1-Invasive duct carcinoma (Not otherwise specified) (NOS). 75%

A) - Scirrhous carcinoma: 70%



Scirrhous carcinoma

In middle aged women.

-*MAC*: Small hard irregular mass with flat undersurface.

Cut section is gritty, grayish and concave.

-*MIC*: Malignant Spheroidal cells arranged in irregular clusters separated by excess dense fibrous stroma.

B) - Atrophic scirrhous carcinoma..... 5%

In elderly (post menopausal) women with atrophied breasts.

-*MAC*: small hard slowly growing mass with late spread (good prognosis).

-*MIC*: The cellular element is minimal with excessive fibrosis.

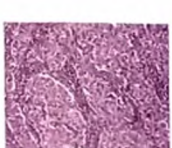
2-Medullary (encephaloid) carcinoma..... 5%

Usually in young adult females with well developed breasts.

-*MAC*: large soft irregular mass.

Cut section is soft, grayish & convex with areas of hage. & necrosis.

-*MIC*: large masses of malignant spheroidal cells separated by scanty vascular stroma with dense lymphocytic infiltration. The cell masses may show central necrosis.



Medullary carcinoma

3-Inflammatory carcinoma (Mastitis carcinomatosa) 2%

- It is regarded as an aggressive subtype of medullary carcinoma.
-Usually occurs during pregnancy and lactation.
- It is rapidly metastasizing with bad prognosis.
-*MAC*: large rapidly growing mass with marked oedema.
-*MIC*: very high cellularity and very little fibrosis.

4- Paget's disease of the breast. .....1 %

Definition: It is malignant erosion of the nipple and followed by intramamary carcinoma.

Aetiology: intraductal carcinoma spreads early to the nipple and areola then late to the gland.

Pathology:

More in ♀ > 40 years

MAC: unilateral, well defined, non-oozing eczema eroding the nipple & areola.

MIC: There are:

- Hypertrophy of all layers of epidermis.
- Paget's cells: clear vacuolated cells with small dark nuclei in the deep layers of the epiderm
- Round cells infiltration of the dermis.

Paget's disease of the breast

5-Mucinous (colloid) carcinoma & Tubular carcinoma 4%

-More in elderly women.

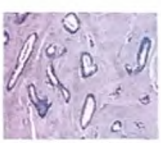
-*MAC*: well defined masses.

-*MIC*: Colloid carcinoma → lakes of mucin between the cells.

-Tubular carcinoma → well, differentiated tubules lined with malignant-cells.

-They have better prognosis than NOS carcinoma.

Mucinous carcinoma



Tubular carcinoma

6-Invasive lobular carcinoma 7%

-It arises from lobules.

-*MAC*, well circumscribed mass.

-*MIC*: small malignant cells arranged in linear pattern *(Indian Files)*.

-Multicentricity & bilaterality should be suspected in lobular carcinoma.

7-Rare cancers..... 1%

a- Invasive papillary carcinoma.

b- Invasive comedo carcinoma.

c- Adenoid cystic carcinoma.

d- Epidermoid carcinoma.

lobular carcinoma

Spread:

1-Local

1-Mammary gland

- Infiltration along milk ducts with fibrosis → nipple retraction.
- Infiltration of Cooper's ligaments → skin dimpling.

2-Deep structures:

Pectoral fascia, pectoral muscles, serratus anterior muscle then to chest wall .

II-Lymphatic spread:

See the anatomy by both lymphatic embolization & permeation.

III-Blood spread:

-To the lung, liver, brain & bone especially vertebrae due to communication between intercostal veins and vertebral plexuses.

-The bone metastases are usually osteolytics ,osteosclerotic or both.

IV-Transcoelomic spread:

-Krukenberg's tumor and nodules in Douglas pouch 2ry to peritoneal implantation.

Complications:

1- Skin ulceration & fungation.

2- Infection.

3- Bleeding per nipple.

4- Spread to deep structure.

A- Chest wall & infiltration of intercostal nerves.

B- Intercostal veins → vertebral plexus→ vertebral metastasis.

C- Pleura → malignant pleural effusion.

5-Lymphoedema of the arm (brawny arm). Due to extensive lymphatic infiltration of the axillary L.N.s. However, it usually develops after surgical axillary clearance.

6-Distant metastases.

Incidence:

-It is the commonest malignancy in females.

-It is commoner in (high risk group):

1- Females with early menarche and late menopause.

2- Spinsters, nulliparra and non-lactating females.

3- Female with positive family or history of cancer of the other breast.

4- Obese females.

5- Female taking contraceptive pills.

6-Patients exposed to heavy irradiation.

Clinical picture:

Symptoms:

1-Mass: in the breast (1ry) or in the axilla (2ry) usually discovered accidentally during bathing

2-Discharge per nipple:

-Bloody in duct carcinoma.

-Paste like in comedo carcinoma.

-It may contain necrotic material.

3-Skin and nipple manifestations:

-Dimpling.

-Fungation and ulceration.

-Nipple retraction and intractable eczema.

4-Symptoms of distant metastases (cough, bony aches.....etc).

Signs

I-General:

Signs of distant metastases (hepatomegaly, ovarian mass, pathological fractures ....etc.)

II-Local:

It depends on the pathological type:

smaller and higher

(1) Duct carcinoma:

-Subareolar mass may be felt.

-Bloody discharge on squeezing.

-It has a good prognosis due to early presentation and late spread.

less protrusion

(2) Scirrhous carcinoma: (the commonest)

The examination includes:

1) The breast: compared to the healthy side.

- It appears smaller and higher
- It protrudes to a lesser extent on leaning forward.
- It ascends to a higher level on raising the arms up.

higher ascent

2) Nipple & areola:

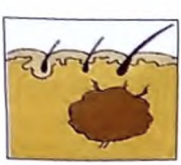
- Change of direction (normally downward, forward & laterally).
- Recent nipple retraction due to fibrosis of milk duct.
- Fixed to a lump.
- Discharge on squeezing.

3) Skin:

-*Dimpling or puckering* due to contraction of Cooper's ligaments.

-*Peu d'orange*: It is thick nonpitting oedema of the skin pitted at sites of hair follicles & sweat glands. It is due to obstruction (compression) of skin lymphatic by the tumor.

-*Skin nodule*: due to lymphatic permeation to the skin.



Peu d'orange

-*Cancer en cuirasse*: due to extensive lymphatic permeation to the skin forming a hard case (like shields of wars).

-*Skin ulceration*: due to infiltration and fungation of the tumour. through the skin

Skin nodules & Cancer encuirasse

4-The mass: characterized by:

-felt easily with both the tips of fingers and their palmer surfaces.

-Hard mass with flat under surface.

-Well circumscribed edge due to the hard mass in a soft fatty tissue.

-Not freely mobile inside the breast (fixed to the breast tissue).

-May be fixed to skin, underlying muscles or even the chest wall.

5-Axilla:

Enlarged axillary LNs. Test consistency, tenderness & mobility.

6-The arm:

Brawny edema.

(3) Atrophic scirrhous: carcinoma:

-Small hard mass in the breast (atrophied) in elderly females.

-It has a very good prognosis (slow growth and late spread).

(4) Medullary (encephaloid) carcinoma:

-Large soft mass in the breast (well developed) in young females.

-It has a good prognosis (early presentation, later spread and young age of the patient)

(5) Mastitis Carcinomatosa:

-Rapidly growing mass in the breast usually in pregnant or lactating females.

-It is accompanied by inflammatory reaction that resembles acute abscess from which it can be differentiated by the following:

1- No fever.

2- The oedema is more extensive (involved > 1/3 of the breast).

3- The skin is dusky red with dilated veins.

4- The axillary L.N.s are hard, fixed and painless.

5- No response to antibiotic therapy.

6- No leucocytosis.

-It has a very bad prognosis (misdiagnosis, aggressive nature, rapid spread).

6-Paget's disease of the breast:

-Malignant eczema eroding the nipple & areola and may be with underlying palpable mass.

-It is differentiated from ordinary eczema by:

Paget's disease	Ordinary eczema
-older female > 45y	-During childbearing period
-Unilateral	-Bilateral
-well defined with erosion of nipple & areola	-ill-defined non erosive
-No ooze, no itching, no vesicles	-oozing, itchy with vesicles
-may be palpable mass	-No masses
-No response to corticosteroid	-responds well to corticosteroid

-It has a good prognosis (early diagnosis and slow growth)

Investigations:

I-Investigations for early detection of cancer breast:

-When these investigations are used for screening, they are localized to high risk groups. They are performed annually and include:

1-Periodic self-examination:

-Each female above the age of 20 years should examine herself infront of a mirror after each menstruation for:

-Change in the size or level of the breast

-Dimpling of the skin, retraction of the nipple.

-Palpable mass in the breast or in the axilla.

2-Soft tissue mammography:

-Using low voltage-high amperage X rays on an ultrasensitive film.

-It demonstrates the soft tissue architecture of the breast and detects small masses.

-Malignant mass appears as dense irregular shadow with fine stippling (micro calcifications) and thickened overlying skin.

Xeromammography: (Xero = dry)

Using a photoconductor that conducts the image on a special selenium plate to give a dry positive film.

Irregular shadow with fine stippling

*Indications of mammography:

1-detection of small subclinical tumour especially in large breasts.

2-differentiation between benign and malignant mass.

3-Screening researches.

3- Breast Ultrasonography:

-It differentiates solid from cystic lesion and localizes masses.

Indications:

1-Localizes masses in radiologically dense breasts.

2-Ultrasonic guided needle biopsy.

4- Magnetic resonance mammography (MRI of the breast):

It is non invasive accurate imaging technique.

II-Investigations to prove malignancy.

1-Fine needle aspiration cytology (FNAC):

Using needle with small caliber to obtain fluidly sample of tissue for cytological examination.

2-Trucut needle biopsy = core needle biopsy:

It obtains core of tissue for standard histopathological exam.

Disadvantages of needle biopsy:

1-Sampling error, better under U/S guide.

2-It needs experience for good interpretation.

3-It is claimed to cause dissemination.

3-Incisional biopsy:

For incompletely resectable tumour to take a piece meal biopsy.

4-Excisional biopsy:

For small questionable lumps → complete excision for HPE.

5- Punch biopsy of skin in Paget's disease

6- Frozen Section biopsy:

To make an immediate (intra-operative) pathological examination.

*If malignant → proceed to radical resection (pre-operative consent of the patient is a must).

7- Exfoliative cytology of nipple discharge:

It is not reliable.

III-Investigations to detect distant spread:

1-Chest X ray.

2-Computerized tomography: brain & abdomen.

3-Ultrasonography: pelviabdominal.

4-Isotope scanning: liver (Tcm^{99}), Bone (F & Tcm^{99}).

IV-Investigations to follow up

1-Tumour markers: 1-(CA 15-3) and Mammary Carcinoma Antigen (MCA)

They are prognostic rather than diagnostic.

2-Isotope scanning.

Triple Assessment is the combination of clinical examination, breast U/S or mammography and fine needle aspiration cytology. It is triple approach to a breast lump, if the three tests are concordant treat as so, and if they are inconcordant proceed for excisional biopsy.

Differential Diagnosis:

1-From other causes of hard mass in the breast (see later).

2-From other causes of bleeding per nipple (see later).

3-Inflammatory carcinoma from acute mastitis (see before).

4-Paget's disease from ordinary eczema (see before).

Clinical Staging

There are two popular systems of staging

1-T.N.M. staging

1-Tumour:

T0 = No palpable tumour.

Tis = Carcinoma in situ [DCIS, LCIS and early Paget's disease (with no mass)].

T1 = Tumour < 2cm with no skin involvement except Paget's disease.

T2 = Tumour 2-5cm.

T3 = Tumour > 5cm

T4 = any size with skin invasion or chest wall fixation

	T1	T2	T3	T4	
N0	I	II	II	IIIB	IV
N1	II	II		IIIB	IV
N2				IIIB	IV
N3	IIIB	IIIB	IIIB	IIIB	IV
	IV	IV	IV	IV	M1

2-Nodes:

N0 = No nodal metastasis.

N1 = Mobile palpable ipsilateral axillary LN(s).

N2 = Fixed palpable ipsilateral axillary LN(s).

N3 = involved internal mammary LN(s).

3-Metastasis:

M0 = no detectable metastasis.

M1 = Distant metastases including involved homolateral supraclavicular LN or contralateral axillary LN(s).

(Skin dimpling or nipple retraction doesn't affect classification).

Manchester staging

Stage I:

-The growth is confined to the breast.

-Skin involvement if present doesn't extend beyond the tumour margin.

Stage II:

The same as stage I + palpable mobile axillary LN(s).

Stage III:

-Skin involved or peu d'orange wider than the tumour, but still limited to the breast.

-Fixation to pectoral muscle, but not to the chest wall.

-Fixed homolateral axillary LN(s).

-Involved homolateral supraclavicular LN(s).

Stage IV:

-Skin involvement wider of the breast including cancer encuirasse.

-Fixation to chest wall. - involvement of the other breast - Distant metastasis..

Treatment

I- Non invasive carcinomas

A. Lobular carcinoma in situ (LCIS)

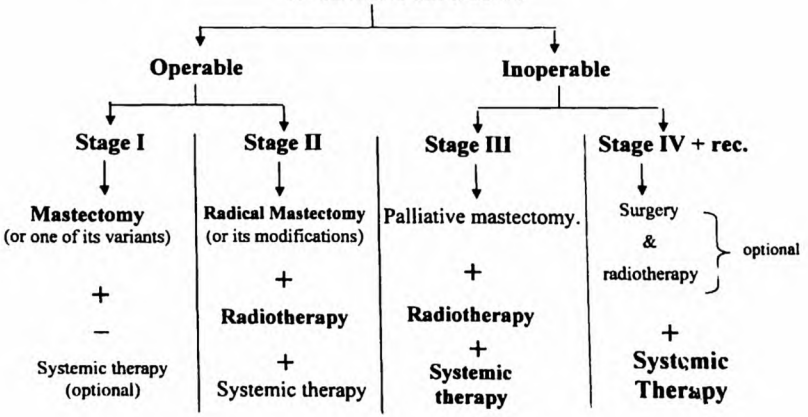
Risk reducing bilateral mastectomies in high risk patients,

Only observation in low risk patients

B. Duct carcinoma in situ (DCIS)

1. Non symptomatic and non palpable (DCIS) (detected by screening mammogram)
   - Local excision with clear excision margins followed by radiotherapy.
   - Mastectomy, the most effective treatment but recently considered overtreatment
2. Symptomatic and palpable (DCIS) are managed as invasive type.

II- Invasive carcinoma



I-Operable cases

A-Stage I:

Treated with radical mastectomy or one of its modifications, the specimen sent for H.P.E. if axillary L.N.s. are not involved, no further treatment is needed but only follow up.

-Nodes positive. cases are treated as stage II.

Recently, systemic therapy may be given to stage I .

Mastectomies

1-Halsted's radical mastectomy:

We remove:

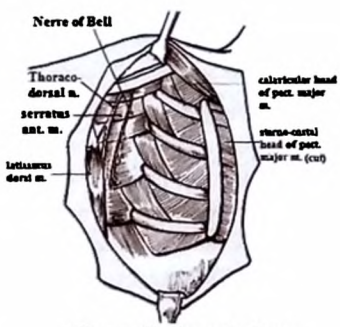
a- The whole breast tissue.

b- Ellipse of skin including area 2 inches around the tumour & nipple areola.

c- Fat & fascia from the clavicle to and including the upper 1/4 of the rectus sheath and from the sternum to latissmus dorsi.

d- The whole fat, fascia & LNs of the axilla.

e-Pectoralis major (sternocostal head) & pectoralis minor ,and interpectoral LNs


Nerve of Bell
Thoraco-dorsal n.
serratus ant. m.
latissmus dorsi m.
clavicular head of pect. major m.
sterno-costal head of pect. major m. (cut)

After radical mastectomy

We should preserve:

1- Axillary vessels, Cephalic vein & nerves of brachial plexus.

2-- Nerve of serratus anterior muscle (*nerve of Bell*) or long thoracic nerve..

3 Nerve to latissmus dorsi or thoracodorsal nerve.

Some surgeons prefer to begin with axillary dissection first to:

* Assess operability.
* Decrease lymphatic spread due to manipulation during mastectomy.
* Decrease the time of chest exposure

2-Extended or super-radical mastectomy of Urban (not accepted)

Radical mastectomy + removal of:

* Homolateral 1/2 of the sternum and costal cartilages from 2nd to 5th.
* Internal mammary LNs.& * Supraclavicular LNs.

-It is rarely done now, as it carries higher morbidity & mortality without improved survival.

3-Modified radical mastectomy of Patey (most popular):

-Radical mastectomy with preservation of pectoralis major.

-The pectoralis minor and the interpectoral L.N.s. are removed.

-It has a lower morbidity with the same survival rate as Halsted radical mastectomy.

4-MC. Whirter's technique:

-Simple mastectomy + heavy postoperative irradiation to axilla, mediastinum and supraclavicular region (3weeks after operation.)

-It has the same results of Halsted's operation.

5-QUART = Quadrantectomy + Axillary dissection + Radiotherapy:

-Removal of the involved quarter of the breast + clearance of axilla + radiotherapy of the remaining breast tissue and remaining LNs. (mediastinum and supraclavicular region).

-It is claimed to have the same results (best in the upper outer one).

6-Lumpectomy + supervoltage Radiotherapy:

-Removal of the lump with surrounding breast tissue (safety margine) + super voltage radiotherapy to the remaining breast and draining LNs.

-Not yet accepted as it ignores the multicentricity of breast carcinoma.

7-Subcutaneous mastectomy with augmentation mammoplasty using breast prosthesis:

It is suitable for very small or in situ tumors

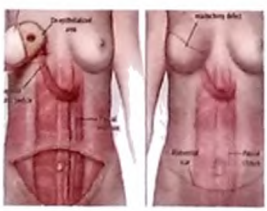
8-Breast reconstruction:

A-Breast prosthesis: after subcutaneous mastectomy or with skin flap.

B- latissimus Dorsi (LD) flap based on the thoracodorsal art. Usually with prosthesis

C- Pedicled Transverse Rectus Abdominis Myocutaneous (TRAM) flap: based on the sup. epigastric artery OR free deep inferior epigasteric artery (DIEP) flap

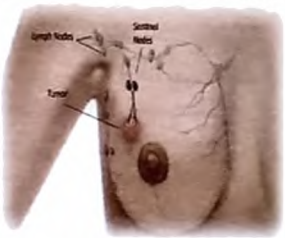
LD flap



TRAM flap

DIEP flap

N.B. Sentinel LN. is the first draining LN. for the tumour. It is known by injecting patent blue dye in the surgical bed after removal of the tumour and tracing the dye after sometime to the axilla. The sentinel LN is discoloured blue. If frozen section exam. of this LN. was free, there is very little possibility that other LNs in the axilla are affected and axillary clearance is not advised. This new approach is called *axillary mapping.*



Sentinel lymph node

B-Stage II

I- Radical mastectomy, or its modifications.

II-Adjuvant Radiotherapy.

A-Pre operative radiotherapy

Indications:

1-Small breasts (e.g. male breast or female with small breast) as they usually skin graft after mastectomy (unsuitable of postoperative radiotherapy).

2-Large tumours: They markedly decrease in size.

Disadvantages:

1-It renders the tissue more vascular.

2-It postpones surgery:

b- Intra-operative radiotherapy:

Radiotherapy to the breast bed after excision of the breast using special apparatus e.g. interbeam.

c- Post operative radiotherapy:

Indications:

1-after radical or modified radical mastectomy with + ve nodes.

2-after conservative breast surgery e.g. MC Whirter, QUART.... etc.

III- Adjuvant Systemic therapy: It includes

A- Adjuvant chemotherapy:

Combination chemotherapy of Cyclophosphamide, Methotrexate and 5 Fluorouracil (CMF) are used for 1 year.

B- Hormonal treatment:

- About 50% of breast carcinoma is hormone responsive i.e. having hormone receptors which stimulate their growth and spread.

- When these tumours are deprived from this hormonal stimulation, they fail to grow and metastasize. Unfortunately, this response is temporary

1- Medical (Drugs):

a. *Tamoxifen*10mg twice daily *or* better *Raloxifen:* antioestrogen. It competes with oestrogen at the cellular level. It causes medical oophrectomy for oestrogen receptor positive cases.

b. *Progestins*: second line drug after Tamoxifen.

c. *Aromatase enz. Inhibitors* e.g. anasterazole. They are new group of drugs that inhibit aromatase enz. which is responsible for oestrogen synthesis in adipose tissue.

d. *Aminoglutethimide*: It is an adrenal suppressant to block adrenal oestrogen. It should be combined with cortisone (medical adrenalectomy)

e. -Androgen & Cortisone.

2- Surgical (Endocrinal ablation):

a. *Oophrectomy:* may benefit Premenopausal or 5 years postmenopausal females

b. *Adrenalectomy* & hypophysectomy (resection or irradiation) are no longer used

Choice of systemic therapy

	Oestrogen receptors +ve	Oestrogen receptor-ve
Premenopausal	Comb. Chemotherapy +Tamoxifen	Combination chemotherapy
Post-menopausal	Tamoxifen alone	Combination chemotherapy

C) Immunotherapy:

Trials to increase immunity by BCG. vaccine, Coll's vaccine, tumour cell vaccine or levimazole

II-Inoperable Cases

A-Stage III

*Palliative mastectomy: to avoid local complications e.g. ulceration and in addition it is psychologically better to the patient

*Radiotherapy: to

1-*The breast*: as an alternative to surgery or as a preoperative RT.

2-*Lymph nodes*: axillary, mediastinal and supraclavicular L.N.s.

*Systemic therapy : as before

B- Stage IV & locally recurrent cases

*Surgery is indicated only in:

-Isolated recurrent deposits specially if ulcerated.

- fungating infected tumours.

*Radiotherapy: is indicated in painful bone deposits.

*Systemic therapy: is the mainstay treatment.

Prognosis:

The prognosis depends on many factors:

1-Clinical staging:

The 10 year survival for clinical stages is:

Stage I: 50% Stage II: 25% Stage III: 5% Stage IV: 0%

2-Pathological exam.:

a-*of the tumours*: inflammatory carcinoma has the worst prognosis.

b-*of the axillary L.N.s.*: (the most important prognostic factor for stage I & II).

3-Site of the tumor:

Cancer in the medial half of the breasts has a worse prognosis.

4-Hormone receptor status:

Hormone receptors positive tumours have better prognosis.

5-Age of the patients:

Worse prognosis in young patient due to stimulatory effect of sex hormones on the tumour.

6-Sex:

Much worse prognosis in males (why?)with rapid spread (local & distant).

Hard Swellings of the breast

Causes = D.D

1-Breast carcinoma (scirrous carcinoma).

2-Fibro adenosis (localized sector type with tense cyst).

3-Hard Fibroadenoma.

4-Chronic nonspecific breast abscess.

5-Traumatic fat necrosis.

6-Plasma cell mastitis (mammary duct ectasia).

Cysts of the breast

I- Cysts from the gland:

1-Cyst of mammary dysphasia [solitary large (Bloodgood) or multiple small].

2-Retention cyst behind a duct papilloma.

3-Galactocele:

*It is retention cyst due to obstruction of milk duct by inspissated milk *during lactation.*

**Fate/comp*: 1-resolution after weaning 2-persistency 3-infection (rare).

**Clinically:* subareolar cystic mass which may extrude milk on pressure.

**Treatment*: - Conservatives for spontaneous resolution.

-Surgical: excision if persists or drainage if infected.

4-Serocystic disease of Brodie.

5-Papillary cystadenoma.

6-Intra-cystic paoillaferous carcinoma.

US picture

II- Cysts from the stroma:

Lymphatic cyst, blood cyst and hydatid cyst.

Treatment:

Aspiration (diagnostic & therapeutic):

Malignancy is suspected if one or more of the following criteria exist:

1- Red aspirate (bloody or sanguineous).

2- Residual mass after complete aspiration.

3- Refilling of the cyst after some while.

4- Revealing malignant cells by cytological examination of the aspirate.

Excision:

Excisional biopsy if any of the above criteria is present.

Discharge per nipple

Causes & types:

1-Milk: a-Lactation b-galactorrhea c-galactocele.

2-Bloody : a-duct papilloma (common). b-duct carcinoma.

3-Serous greenish, brownish: in mammary dysplasia.

4-Greenish creamy: mammary duct ectasia.

5-Necrotic: carcinoma.

6-Purulent: very rare in breast abscess.

Cone excision

Investigations:

1-mammography: to detect impalpable lumps.

2-Localization test: by differential pressure to localize the offending duct(s).

3-Ductography: to diagnose duct papilloma & carcinoma.

4-Benzidine test: to detect occult bl. in the discharge.

Treatment:

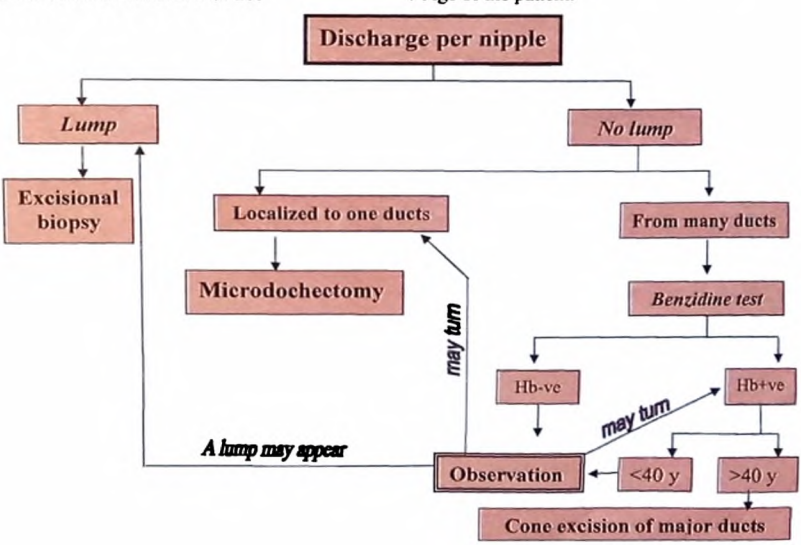
It depends on the following factors:

1-Associated lump or not

2-Localization to one duct or multiple ducts.

3-Presence of occult bl. or not

4-Age of the patient.



Diseases of the male breast

1-Mastitis of puberty

2-Traumatic mastitis

3-Gynaecomastia

Definition

It is enlargement of the male breast gland to simulate of a female

Aetiology: = causes = types

Primary = physiological = Idiopathic: (The commonest)

1-Neonatal gynaecomastia duo to maternal sex hormones

2-Pubertal gynaecomastia: common usually resolved within 2 years

3-Senile gynaecomastia: due to reduction of tests

Secondary gynaecomastia:

1-Congenital: Klinefelter's syndrome, anorchism

2-Infection: bilateral epididymo-orchitis

3-Inflammatory: leprosy may cause bilateral testicular atrophy

4-Metabolic: liver cell failure due to decrease oestrogen

5-Iatrogenic:

A-Drugs: - Aldactone Brenerdine (reserpine) and Cimetidine Digitalis.

-Oestrogen therapy for prostatic carcinoma

B-Surgical: after castration

6-Neoplastic: Testicular teratoma, adrenal tumours, and bronchogenic carcinoma.

Treatment

-Idiopathic type: usually resolves spontaneously within 2 years

Persistent gynaecomastia→ subcutaneous mastectomy

-Secondary type: treatment of the cause

4-Mammary dysplasia: not rare

5-Fibro adenoma not rare

6- Carcinoma

-It represents only 1% of all cancer breast

-Because of deficient breast tissue fixation, ulceration & distant spread is usually early (worse prognosis inspite of earlier diagnosis)

-Staging & treatment are as in female but castration gives better response with a remission about 30 months in 70% of cases. Tamoxifen gives good results and replaces orchidectomy.

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